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CONGENITAL PYLORIC SPASM AND CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS IN INFANCY.

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There is no disease of infancy and childhood which, in the past five years, has attracted more attention than the so-called congenital stenosis of the pylorus, or, as it was formerly and is still called, congenital spasm of the pylorus. New methods of diagnosis, and the bad results of feeding in these cases seem to have brought about a more frequent recognition of the condition than formerly. When Beardsly described his case very little was thought of it, and apparently the condition was allowed to pass unrecognized. The publication of Landerer, in 1879, and that of Maier, in 1885, caused renewed discussion of this condition. Landerer described some autopsies in adult subjects, some of them having lived until the forties, which showed postmortem a narrowing of the pyloric orifice of the stomach. The history of these cases showed that they had suffered from infancy and childhood and during adult life from the effects of this stenosis.

Hirschsprung, in 1887, communicated two cases of pyloric stenosis occurring in infancy, both of which were fatal, the pylorus in both being considerably thickened, to the extent of not permitting even a small sound to be passed. In one case the pylorus resembled a tumor, and an examination showed a thickening of the longitudinal as well as the circular muscular fibers. Since this time many additional cases of this condition have been reported, so that I am convinced it can no longer be classed among the rare disorders occurring in infancy.

With all the observers who have seen these cases and discoursed upon them, there has arisen a difference of opinion which is not only confusing but disquieting. Some have criticised the observing capacities of others who have published their cases, and discussions have become almost personal in their nature. Especially violent was the discussion after the publication of the surgeon Stern, who was the first to operate in these cases when they appeared to him to be hopeless in their nature. The discussion of his case and the interchange of published letters between the surgeon and Pfaundler are now a matter of record. I shall not enter into this part of the subject, but simply state that Pfaundler's position can be explained by a possible difference in material.

I shall endeavor to show in this paper that this is a disease which is rather vacillating at times in its symptomatology; that the men who insist that the patients get well spontaneously are correct when their material is analyzed, and that those who insist on operative procedures in certain cases might be correct if their material is analyzed in a like manner. Both parties are possibly correct, but they have both been looking at opposite sides of the shield. It is for this purpose that I have decided to publish my own cases and critically analyze them in detail with an endeavor to introduce some clearness into the subject obscured by useless, and to me purely academic, discussions.

I may be pardoned if, for the sake of completeness, I go lightly into the symptomatology of an affection which has been so widely considered. As a rule, in cases of spasm or congenital stenosis of the pylorus the children are born perfectly well; the great majority of them have been breast-fed; I should say from the literature that fully two-thirds of the children were breast fed from the start. After a while, varying from one to four days, in other cases seven days, and in a great many cases the third week after birth, in exceptional cases the eighth week after birth, the vomiting begins. In a few cases, as in some of my own, there is a history that an attempt was made to feed the baby, in addition to the breast, on the bottle, and in these cases the vomiting began from the attempts at mixed feeding. In other cases there is no such history, the mother's milk being the only food; the milk seemed to be abundant, and there was no change in the milk or in the mother to account for the disturbance in the child. The vomiting occurs at first at intervals throughout the twenty-four hours, and soon becomes persistent, the child rejecting sometimes a portion or all of every nursing. Sometimes the history will show that the infant has rejected more than it had taken. This is quite characteristic, and points toward a retention in the stomach of some of the previous feedings. With the vomiting there is a steady emaciation or a stationary weight. If the weight is stationary, the patient is fortunate. If the emaciation is progressive, we see in a few weeks an infant which had been perfectly well at

birth, weighing the normal or above the normal, reduced to a distinctly marantic condition. With the vomiting there are other signs of constitutional disturbance. It seems that every time the breast is given to the child, or within a few minutes after nursing, there are evidences in some cases of pain; the children will cry, and this will be told spontaneously to the physician by the mother. In addition there is constipation in most cases, or the movements are small, minimal in quantity, sometimes fluid in consistency, or they may be greenish. As a rule, the movements indicate that very little has passed through the gut. Physical examination in these cases reveals in the vast majority of instances a characteristic condition of the surface of the abdomen. On the introduction of food there is a peristalsis visible to a greater or less extent on the surface of the abdomen. This peristalsis begins underneath the left costal border, passes forward to Traube's triangle, and there seems to stop, being interrupted by a sort of groove, and is taken up again by a second wave of peristalsis which passes onward beyond the ensiform cartilage, then downward, and disappears. Some authors have described a reverse peristalsis just previous to vomiting, but I could never convince myself of the fact, possibly because I have not seen these particular cases. If there is a reverse wave of peristalsis, it must be instantaneous, and I have not yet observed it. Ibrahim also expresses his lack of information on this reverse wave. In some cases I have seen the peristalsis so extreme that just previous to vomiting the stomach would in a manner erect itself on the abdomen and divide itself distinctly from what appears to be the pyloric end of the stomach; it would contract, and then the vomiting would take place.

The vomiting is projectile in its nature, as if there was a sudden violent contraction of the stomach and a forcing upward of the contents. In some cases careful examination during this period of contraction and peristalsis reveals a small hard nodule, cartilage-like in consistency, situated sometimes beneath the liver or its border and running directly downward toward the umbilicus. This structure, situated deeply against the vertebral column, is undoubtedly the pyloric end or valve of the stomach as it meets the duodenum.

Some authors like Ibrahim have described singultus in these cases, and also eructations of gas, but inasmuch as these are quite common in healthy breast-fed infants, it seems to obscure the picture by laying any stress upon them.

The peristalsis which I have described is present in a majority of cases, but it is not necessarily an accompaniment of all of them. It is sometimes entirely absent during the height of the affection, and is only seen at times. The pylorus also may not be palpable, and may not be felt at times; I shall return to these later to show that neither are nece sarily present, but if they exist, they are

pathognomonic of certain cases of the affection. As to the peristalsis, we must be very careful also how we conclude as to its presence or absence, and this I shall also take up later for consideration. I will simply mention here a normal mild form of peristalsis seen in emaciated infants, which must not be confounded with the violent peristalsis I have described as present in some forms of this affection. Some of the most violent cases of vomiting with spasm or congenital stenosis of the pylorus have passed through my hands without the detection of the situation of the pylorus.

Some observers have endeavored to develop peristalsis by snapping the abdominal surface with the finger, or by palpating the stomach and causing the so-called "platsch" noise, or lapping sound, formed by fluid in the stomach. This I also regard as fruitless in considering the symptomatology of these cases.

Inasmuch as I intend to come back to the individual symptoms, I will pass on to a general consideration of other symptoms in these cases. There is no fever. The children, after having nursed, vomit, and, strange to say, will retain fluid which is given them immediately after vomiting. Some authors have been insistent on the physical signs revealed by percussion of the stomach, and have added dilatation of the stomach to the other physical signs in these cases. I must agree with Pfaundler and others that dilatation in the normal or abnormal infant is very difficult to establish, and it is questionable whether it can be proved. The stomach is so subject to variation in size that a percussion of the stomach will reveal very little as to its actual anatomical condition, so that a dependence on signs of dilatation of the stomach as proving the condition which we are discussing is of questionable utility.

I will take up the question as to whether in these cases there is a spasm or a distinct stricture of the pylorus or stenosis. I believe that there is a certain set of these cases, the subsequent history of which proves distinctly that there is at a certain period of the illness a spasm of the pylorus and stomach and at its pyloric end; that for certain reasons this spasm subsides and under favorable conditions the children cease to exhibit further symptoms of their condition, and recover. In other cases, in addition to this spasm of the pylorus there is added also a distinct anatomical condition in which the structure of the pylorus is abnormal; the muscular coats as well as the mucous membrane are thickened and thrown into folds, causing a narrowing of the orifice of the pylorus. This, with the spasm superadded, causes a distinct stricture at this point, which in some cases is extreme. In a great many cases, even this anatomical condition sometimes admits of improvement by relaxation of the stricture, cessation of the spastic condition at the pyloric end of the stomach and opening up of the lumen of the pylorus, and a continued improvement of the patient. In other cases this spasm of the pylorus may subside, but the lumen of the pylorus remains

narrow, and still the children may improve with correct diet. n other words, if the anatomical condition of narrowing of the pylorus is not complicated any longer with a spasm, which exaggerates this condition, a certain amount of food is allowed to pass through the narrow orifice into the intestine, is absorbed, and the children improve.

In this way only can I reconcile all the conflicting theories of this condition to be found in the literature. For example, Thompson, who published some of the first cases of this condition, insists that a muscular hypertrophy is superinduced by disordered coördination and contracture of the muscular coats of the stomach, including that of the pyloric end. This incoördinate contraction is initiated by the swallowing at birth of an irritating substance, such as liquor amnii. Its effects on the mucous membrane of the stomach itself are not at first apparent. After a few days or weeks it becomes apparent, and then the vomiting sets in. The overaction of the muscular coats of the stomach, including that of the pyloric end, are a direct cause of hypertrophy of the coats of the stomach. In other words, a form of functional hypertrophy due to overwork of the stomach develops.

Another theory which can only be reconciled by the peculiar set of cases seen by the observer, is that of Pfaundler, who insists that all of these cases are spasm cases, and the so-called new tissues or the hypertrophied pylorus, in some of the cases amounting to a distinct tumor, are the result of operative or postmortem contraction, and that such a contracture of the pylorus can be demonstrated at the autopsy table in most infants. I can only say that I feel that possibly Pfaundler had not seen at the time he ventured this theory some of the most aggravated cases of this condition, in which, during life, a hard unmistakable nodular pylorus can be felt through the abdominal wall. The question of postmortem contracture can scarcely enter into these cases, nor can operative contracture.

The third theory is that some of the subjects are born with a congenital anomaly at the pyloric end of the stomach, consisting of a form of newgrowth or hypertrophy of the muscular fibers that go to form the pyloric valve.

I will endeavor to show that all of these theories are in a sense possibly true of many of the cases published. I therefore will divide my subject into those cases in which we can only show from the clinical history and subsequent course of the case that there has been a distinct condition of spasm not only of the pyloric end of the stomach, but at the pyloric valve; and other cases, in which we can say from a study of the clinical symptoms that there is a congenital hypertrophy at the pyloric end of the stomach of the muscular fibers entering into the structure, in some cases so extreme as to indicate a narrowing of the lumen of the pylorus, a stenosis more or less marked, varying in different cases. Even such cases may make a

recovery or a partial recovery, and other cases, apparently exactly similar, will not, according to the opinion of some authors, allow of such recovery without operative interference.

The first set of cases which I wish to report and analyze are those in which there is an undoubted spasm of the pylorus and the pyloric valvular orifice, initiated, as it seems, by some error in feeding the child, by some toxic element which immediately after birth has irritated the stomach itself, causing a mild form of increased peristalsis; these become more disordered and violent, resulting eventually in the symptoms of spasm of the pylorus. It may be that in these cases there is a distinct contracture of the pyloric valve which may or may not be palpable. As a rule, it is not palpable through the abdominal wall.

In some of these cases peristalsis is not evident on the abdominal wall; on the contrary, in some of my cases it was absent. If there was peristalsis, it was of the *fruste* variety; that is, it was abortive. Only a very mild form is seen on the surface. In spite of this, the children vomit, and vomit not only after every nursing, but at times reject even more than is taken, on account of the peculiar condition of the pyloric valve, which does not allow the stomach to be completely evacuated in time for the subsequent nursings. In these cases also, there is, as in the so-called congenital hypertrophic stenosis, a residual amount of food in the stomach between nursings.

The outlook in these cases for the most part is very good. These are the cases which we see so frequently recorded in the literature as having made a recovery without operative procedure. It is surprising how many of them come under the notice of the physician who is especially interested in this line of work. The children in all of these cases are atrophic, or they have remained stationary in weight. Some even show a certain amount of nervous hypertension; they are easily frightened. When undressed and handled carelessly, they start to cry, as if frightened. Thus, as with the stomach, the whole nervous system seems to be in a condition of hyperacusis.

The first case of this kind is one which seems to me to be typical of this variety:

Case I.—Female child, aged five weeks, one of two children, the other child having disease of the spinal vertebræ. The mother has never had any miscarriages, and is well apparently, but the father has a healed Pott's disease. The child is on the breast; it has had no previous illness. The present illness began nine days after birth when the patient began to vomit after each nursing. At first she vomited after every nursing, and then after every other nursing, varying from time to time. The vomit consisted not only at times of all the child had taken in, as the father practically illustrated to me, inasmuch as he collected all that the child had vomited, sometimes four ounces of the breast feeding, but it would vomit

after nursing more than it had taken in; and being a very intelligent man, he surmised that the infant had residual milk in the stomach, because when the child vomited the residual milk was curdled, and when it vomited at times when there was no residual milk there were no curds in the vomit.

The movements, scarcely deserving the name, were fluid, sometimes green and sometimes yellow. The child weighed five pounds thirteen ounces and was in poor atrophic condition. Examination of the other organs of the body, aside from the stomach, which I have mentioned, were negative.

No peristalsis could be seen on the surface of the abdomen, but the pylorus could be felt underneath the liver as a nodular mass, not hard, as in other cases I shall mention, but rather soft and cord-like. When the infant vomited, the abdominal muscles were rigid. The baby would nurse, after nursing would cry; there would be a contraction of the abdominal muscles, and the nursing was expelled. The vomited matter consisted of what the child had nursed, and when seen by me in my office contained a glairy mucus.

Examination of the mother's milk showed that it was very abundant, but at the fifth week it had a peculiar dirty appearance, so that evidently, from a gross physical examination, it was unsuitable for the baby.

This patient was sent to my hospital service and I had full opportunity to observe it. As soon as the child was taken away from the mother's breast it was put on a feeding at first of simple water, and was fed somatose by the rectum for twenty-four hours. The simple water contained a little citrate of sodium. After twenty-four hours the infant was placed on an ordinary percentage mixture of top milk, and each nursing was accompanied by a powder of citrate of sodium. The vomiting ceased as if by magic, but the child, in spite of variations of percentage of fat and proteids to within a limited period, refused to increase in weight on cows' milk mixture, and was put on a combination of the breast (wet nurse) and the bottle, when it increased slightly in weight. After it had been in the hospital for two weeks it was removed on account of an outbreak of measles in the ward and was sent home. It had vomited but twice in two weeks in the hospital. When at home, outside of my strict watch, on a wet nurse, it vomited only once a day, and did well. Having gained, the wet nurse was changed for economical reasons. The patient began to vomit again. The milk analysis showing 5 per cent. of fat. A change of wet-nurse again resulted in a cessation of vomiting. The infant is now thriving.

Discussion. This is a case in which we can say in all likelihood that the mother's milk was the direct cause of the child's vomiting, as also a subsequent wet nurse's milk. The mother states that after the eighth day the child was given in addition the bottle, and the vomiting followed. This administration of the extra bottle of milk, it appears

to me, with the irritating qualities possibly of the mother's milk, or the high fat percentage of the wet nurse's milk, have been responsible for the spasm of this stomach, which was of a purely toxic type. This case can scarcely be confounded with the so-called habitual vomiting of infants, which is quite frequent in practice. This child vomited to such an extent as absolutely not to retain anything on its stomach after the nursing, and at times only retained enough to ferment in the stomach and be evacuated at the next nursing. It had been under the care of a distinguished pediatrist, who had put it on dionin, without results.

Unfortunately we do not know the initial weight of this baby, but the mother states it was a plump baby when born. I feel that its future will be good, inasmuch as it has improved on the breast nursing.

Case II.—The second case is a typical one of spasm of the pylorus, which came to my service at the Mt. Sinai Hospital. Female infant, aged seven months. The mother has tuberculosis and Bright's disease; otherwise the history is negative. One of her children died of meningitis at the age of eighteen months, the other is still living and well. This baby was born after an easy labor, but was never breast fed. It was fed on condensed milk and water for two weeks; it was in the Children's Hospital in Albany from August to October, three months before I saw it. At that time the child had diarrhœa, watery stools every day, sometimes green, sometimes mixed with mucus. The infant vomited almost every feeding. The vomiting consisted at that time of cheesy, sour smelling material. It never vomited blood, nor passed blood by the rectum; there was no cough. The child was admitted to the hospital, in fact, for this vomiting, although there are no data as to exactly when the vomiting began. When admitted to my service the child had general furunculosis; it was in miserable condition; it was restless, cried a great deal, did not sleep. For the past month the child has vomited daily; it vomited every feeding cheesy, sour-smelling material. The bowels were constipated, the child was losing in weight, and was extremely atrophic. When admitted to the service the child weighed seven pounds and nine ounces. It is not necessary to give the physical examination on admission as it does not bear on what has been noted.

In this case I studied the abdomen carefully after having first watched for peristalsis, but failed to get it in the empty condition of the stomach. When the feeding was given to the child, the stomach was studied. There was a very mild form of peristalsis present, and in spite of this no pylorus could be felt distinctly. The child was admitted January 5, and up to January 22, her weight ran down to six pounds and eleven ounces. She vomited practically everything that was given to her. Forced feeding was tried, and in this forced feeding at first citrate of sodium was added to

the milk. From the time of the forced feeding the spasmodic vomiting ceased, and was replaced by an occasional vomiting, until finally on January 26, when the citrate of sodium was given, the frequency of the vomiting dwindled to once a day, and after February 1 the infant practically did not vomit at all.

Discussion. This is a case which was closely watched. Out of eight feedings a day, at first the infant vomited from six to eight without any abdominal symptoms, and with a gradual diminution in weight. When the vomiting ceased the weight gradually increased until its discharge from the hospital, exactly one and one-half months after admission, weighing eight pounds and five ounces, an increase of two pounds over the lowest weight, that is, January 22, having been discharged February 16. This is a typical case of spasm of the pylorus without any symptoms of hypertrophy of the pyloric end of the stomach, and no marked peristalsis, in which forced feeding was resorted to for a few days, followed by the ordinary nursing, with an increase of weight and a tolerable cure of the condition. This case was under observation at times for the purpose of deciding whether operative interference would be necessary. The course of the case speaks for itself.

Another exquisite example of simple spasm of the pylorus without marked hypertrophy and possibly no great stenosis of the valve is the following case:

CASE III.—Male, aged four weeks, the only child. The mother has had no miscarriages, she and the child both being healthy. It was a forceps delivery; the child, weighing eight pounds at birth, was breast fed, had gained every day on the mother's breast until two weeks after birth, when vomiting began. The child vomited after every nursing for ten days previous to being seen by me. The mother allowed it to nurse as long as it pleased, she having plenty of milk, and her breasts at times caked. It vomited after every nursing sour, yellowish material of great quantity. The child had moderately large movements, which were greenish. When brought to me it weighed seven pounds and five ounces, was poorly nourished, atrophic in its condition. Physical examination was negative, and when observed it vomited during nursing, but when nursed again would retain its food. At the first examination there was no peristalsis and no pylorus could be felt. The vomited matter was large in amount; vomiting occurred almost immediately after nursing, and was projectile. The mother states that having vomited the baby will retain a second nursing quite well for at least half an hour. The mother's milk seems to be good in quality and color. I noticed, however, that the child had a traumatic stomatitis; its mouth had been washed and treated roughly.

In order to test whether the breast or the bottle was at fault, I placed the child on alternate breast and bottle feedings. I found that it vomited the breast more than the bottle, therefore I

stopped the breast entirely and placed it on the bottle. When seen a week subsequent to the first visit, there was a slight *fruste* peristalsis noticeable on the surface of the abdomen, but no pylorus could be felt. The vomiting continued after almost every feeding, the weight diminished to seven pounds four ounces, and still no peristalsis could be seen, but the pylorus could be felt under the liver in the neighborhood of the umbilicus like a thin cord-like structure. The child continued to vomit. After three weeks of observation it had diminished in weight to six pounds and fourteen ounces. The pylorus could then be felt as large as a bean underneath the liver half way between the umbilicus and the free border of the ribs. Whenever the child has a spasm of the stomach it cries as though in pain, and is only relieved when the vomiting occurs. In spite of loss in weight the infant looks well. The child is extremely nervous, hypersensitive, and when the spasm sets in the child starts, and then the vomiting occurs. After three and one-half weeks of vomiting the child was put on a wet nurse, and began to gain gradually. The wet nurse was changed, and it was then put on a very good breast, and the vomiting gradually ceased, taking place only once a day, and the child is doing well. It gained fully two pounds in one month of the new nursing.

Discussion. This is a case of clinical spasm of the pylorus which was closely watched for a month with the idea of possible surgical interference on account of steady loss of weight. The friends of the infant, including the mother, were intelligent people, understood the situation exactly, and coöperated with the observer. There was no question as to the vomiting; every particle of food taken into the stomach was rejected from the time of the observation until the infant was placed on a breast which was as nearly perfect as possible. The mother's milk evidently was responsible in this case for the initial vomiting, for as soon as the infant was relieved partly of the breast, this leading feature of the case subsided with the bottle feedings, although not completely so, and continued unabated with the breast. When the mother's breast was completely suspended improvement in the vomiting occurred, but the weight was rather stationary, if the child did not lose. With the ideal breast the vomiting ceased almost entirely, taking place only once in twenty-four hours.

Here are three cases of aggravated spasm of the pylorus, which I am assured would come under the classes, so frequently mentioned by Heubner, Pfaundler, and others, who insist that they can get well without operation. I feel also assured that possibly others, who have not had so many of these cases come under their observation, and who did not have the opportunity to observe them for the length of time that I had before an improvement set in, might have resorted to operation; not that I think operation would have harmed any of them; on the contrary, it might have resulted in

the infant's having been relieved of the spasm and improved more rapidly than with the preceding methods; but they illustrate perfectly how cases of simple spasm will not only improve and get well, but may at times be mistaken for another set of cases, which are still more aggravated in their symptomatology.

I will now take up the study of cases from my records of congenital spasm of pylorus, which at first showed symptoms fully as severe as those which have just been noted, but which improved rapidly under observation, or having passed from observation showed after many accidents and much experimentation with diverse forms of feeding, loss of weight to a most desperate degree, according to the statement of the attending physician. They then improved spontaneously on foods, which we as scientific men consider most unsuitable.

CASE IV.—R. O., male, native of the United States, aged six weeks, patient of Dr. S. The first child; mother never had any miscarriages; father and mother are in good health. At birth baby weighed seven pounds. When it came under observation the infant was fed partly on the breast and partly on the bottle. It had vomited since birth, and having vomited the breast, was taken off the breast and placed on an artificial feeding. The vomited matter was watery and acid; the baby had gained in spite of vomiting to a slight degree. At times it does not vomit at all; at others it vomits all that it has taken. The bowels have a good color, but the infant is constipated. The main food now is the breast, the mother having gone back to it after several vain attempts with the bottle. The weight of the infant is seven pounds and seven ounces; it is in tolerably good condition, although atrophic. The physical examination is negative, and a study of the abdomen gives no peristaliss. Diagnosis: spasm of the pylorus.

The infant having been taken off the breast and placed on a substitute feeding, did not improve, and disappeared from observation, having tried no less than three different kinds of foods within two months. It vomited everything, the vomiting having increased since the first visit, a period of two months; spits up, if it does not vomit. Vomits a little at each feeding, and vomits very severely twice a day all that it takes in. After another week of attempted feeding with foods which I shall not mention here, the vomiting continued about after each feeding. After an interval of about two weeks I saw the child again, and the vomiting continued. The increase in weight in two weeks was one-quarter of a pound. The child then disappeared from observation. The parents were very much opposed to placing the child on a wet nurse, and seemed rather determined to have the child die rather than resort to that method of feeding, having peculiar views on the subject. The child continued for two months, and lost in weight from the time I had first seen it to such an extent that the attending physician had made up

his mind that operative procedure would be necessary. He then placed it on a patent food, and strange to say, from the moment the trial of a new food was made, which was simply one of probably dozens which had been tried, the child stopped vomiting and began to gain in weight. Lately I had occasion to see the child, and I have never seen a handsomer baby than the one brought up on what I have always considered, and still consider, a very unsatisfactory food. The baby was eight months old when it was recently presented to me and weighed eighteen and one-quarter pounds, had a rosy hue, was firm in its muscles, and seemed to be perfectly happy. This case never presented any peristalsis, nor could I at any time feel the pylorus.

Case V.—Edith O., aged three months, the only child, one child having died after circumcision; parental history negative; four weeks on the breast, and then was placed on a top milk mixture. It vomits after every feeding either one-half or all that it has taken in; the movements are constipated, small, dark, and undigested. The vomiting began four weeks after birth, when the child was taken from the breast and placed on the artificial feeding. The initial weight at three months was eight pounds and ten ounces. Its general condition was negative. I found no peristalsis, nor could I feel the pylorus. The child was put on an artificial food mixture and did well; although the vomiting continued to a mild degree, it continued to gain on the new food, but very slowly. It remained under observation until it was eight months old, when it weighed eighteen pounds. When fourteen months old it was brought again for observation, and weighed twenty-two pounds and eight ounces, and was in excellent condition.

Case VI.—Harold G., aged six weeks, the first child; parental history negative; was brought up from birth on the bottle; has vomited since two weeks after birth after each feeding, not very much, but "considerable," as the mother puts it. It has vomited all that it has taken, but it is difficult to determine how much. The weight is eight pounds and five ounces, its general condition is good; the slightly contracted pylorus could be felt; there is no peristalsis. A diagnosis of spasm of the pylorus was made. Given a new food, the infant continued to vomit, but gained slowly, and during the entire period of observation the child continued to vomit, but gained steadily, so that after being under observation for six months it had gained nine pounds, and after this continued to thrive. At nine months of age it weighed seventeen and one-half pounds.

Case VII.—Male infant, aged two months, patient of Dr. O. One of two living children; parental history negative. The child was given the breast at first for three weeks, and is now taking a Walker-Gordon percentage mixture. The baby has vomited since birth, sometimes a half or three-quarters of an hour after feeding

it vomits all of the feeding: the milk is curdled, but in spite of this it has gained since birth three pounds. The movements are green, and it has several passages a day. The child suffers from considerable flatus. The weight is ten pounds and two ounces; the general condition is good; physical examination negative, except there is distinct peristalsis visible on the abdomen after feeding, and the contracted pylorus can be distinctly felt. This baby, after considerable coaxing and the addition of sodium citrate to the food, so improved that the vomiting ceased gradually; the child continued to gain, and remained a healthy child free from vomiting.

Case VIII.—Female infant, aged four months, one of two children; parental history negative. Was given the breast a few weeks, and then was put on the breast and bottle. It has vomited since birth nearly the whole of every feeding. Gets the breast twice a day, and for the remainder of the feedings a top milk mixture. It vomits after every feeding nearly all it takes in. The bowels are fairly good, one or two movements a day, has no colic; has only gained four ounces in two weeks. The initial weight at birth is not given, but when seen it weighed eleven pounds and ten ounces, and, although atrophic, was in good general condition. Physical examination negative. There is no pylorus to be felt, although there was an indefinite soft band underneath the liver. A diagnosis of spasm of the pylorus was made. This child was under observation for three months, during all of which time it vomited, but it gained slowly, and at the end of the three months it weighed fifteen pounds (seven months old). It still vomited, but only about a tablespoonful of each feeding. It then passed from observation presumably doing well.

Case IX.—Female infant, aged six weeks, patient of Dr. H.; the only child; parental history negative; weight at birth not given; fed on the breast two weeks, and then was put on a mixture of patent food and milk, although it was given condensed milk and later percentage Walker-Gordon mixture. The baby vomited, though not at first on the breast. Toward the end of two weeks of life it began to vomit at every feeding; had many movements of a watery character, some colic at times. On the percentage mixture it was constipated, and then, through some milk poisoning it had many movements. At the first visit it vomited every bottle and two or three times a day almost all of the feeding. On close observation, slight peristalsis after feeding, and the child seems to suffer after taking the food. There is no palpable pylorus. A diagnosis of spasm of the pylorus was made. This infant came under observation with a weight of six and one-quarter pounds. In a month it gained one and one-half pounds; under careful feeding the vomiting ceased, although at first it continued; the movements were regular, and at times it would have a diarrhœa. It remained one month under observation. The

subsequent history is that the child did very well and is continuing to improve.

CASE X.—This case is one which I saw a number of years ago, and for that reason the history is only partial. It was an only child, female, aged seven months, fed on the breast for a few weeks and then had sterilized milk, peptonized milk, and finally was put on sterilized milk, two-thirds to one-third water. Is very much constipated, rejects a certain amount of several bottles, and vomits a great deal twice a day. The diagnosis at that time was pyloric spasm. Under an appropriate feeding the child continued to improve, but vomited under all feedings, although not so much as it did at first. It was under observation fully a year. At the end of that time it weighed twenty-five pounds, and was a well-nourished child.

It will be seen from what I have just recorded that there are varying degrees of spasm of the pylorus, some of them exceedingly severe, so as to place one in doubt as to the proper therapeutic procedure, and cause great anxiety as to whether any kind of feeding will agree with the child. To show to what extent the process of waiting may be resorted to in these cases, I may, in closing the chapter on the spasm of the pylorus, record a case which was celebrated in its day.

CASE XI.—This child at the age of seven months weighed eleven pounds; an only child; being at first on the mother's breast, began vomiting shortly after birth. The physician in charge placed the infant on a wet nurse. The vomiting became more severe, the child restless at times, to such an extent as to rob it of its sleep. It vomited everything that was given to it. In spite of this the infant was carried along to the seventh month, and almost every physician in New York with any knowledge in feeding had seen the child, and one eminent pediatrist had advised operation as a last resort. When I first saw this child it was exceedingly atrophic; it vomited every bottle that was given it, and almost every kind of feeding known to science and empiricism had been tried. It not only failed to gain, but failed to retain its food.

I was called in simply because the parents absolutely refused to entertain an operation until they had tried one more resort. Physical examination was negative; no pylorus felt; there was no peristalsis. As in many of these infants the abdomen was rigid, and when the infant nursed the bottle, immediately afterward, there was great restlessness, evidences of pain, and the food was rejected. In spite of this the infant held its own at eleven pounds, at seven months of age. It was constipated, the movements were very small. I made a diagnosis of spasm of the pylorus and temporized for a week on an artificial food, which was also rejected. I then suggested a wet nurse, and was informed by every one connected with the case that I had not been called in for that purpose, but

rather for the purpose of trying to give the child an artificial food. I enlisted the attending physician's aid, and promised to obtain a wet nurse for him if he would place the infant at the breast. He promised to do this. From the moment the infant was placed at the breast it commenced to gain in weight, the vomiting gradually ceased and disappeared. The change was simply miraculous. At the age of fourteen months the baby weighed twenty-three and one-half pounds, and was a perfect infant to look at.

This is a case which I think was spasm of the pylorus, in which the diagnosis of congenital pyloric stenosis was made, and in which operation had been advised and held out to the parents as a last resort by physicians whose skill in diagnosis and in treatment of infants is undoubted. But this infant acted like most infants with this affection, it strangely began to improve on the proper feeding, and doubled its weight after seven months.

I hope I have recorded enough cases to show from my experience there is such a thing as *spasm* of the pylorus pure and simple, resulting in constant vomiting and stationary or diminished weight, and a certain amount of suffering to the infant, some infants threatening to pass out of existence unless aided by some heroic measure, such as operation, but all of them improving under the steady prodding of scientific and intelligent feeding. As I have before stated, some of these cases might have been operated upon with a certain amount of justice, and yet I could not find reason for such interference, at least from personal observation at certain stages of the illness. I was fortunate to hold the patients to the last minute until improvement set in, and I have been able thus to demonstrate the practicability of waiting and trying in these cases.

I shall now pass on to the consideration of a condition which I think is an exaggerated state of what has been just pictured, in which not only spasm of the pyloric orifice exists, as well as a spasmodic contraction of the pyloric end of the stomach and the cardiac portion, but in which there is a distinct hypertrophy of the pyloric valve and its structures, a narrowing of the lumen of the pylorus to such an extent as to constitute a distinct growth of new tissues, which can be demonstrated by palpation of the abdomen, and certain other symptoms. In these cases there is a distinct and disordered contraction of the stomach, which is visible on the surface of the abdomen. The peristalsis, so-called, of the stomach is so marked that in the severe cases the stomach seems to erect itself, that is, the cardiac end upon the pyloric end, as if the cardiac end of the stomach was making a supreme effort to drive the food through the contracted pylorus. In these cases the pylorus can be felt distinctly as a hard nodular mass underneath the liver, sometimes midway between the umbilicus and the free border of the liver.

The theory in these cases is familiar from the literature, and it

is, that we either have, according to Thompson, an hypertrophy acquired through the disordered contraction of the stomach, or a congenital condition originating in the uterus and manifesting itself after birth. Under this latter theory we would have a sort of new growth replacing the normal structures at the pylorus.

The third theory, that of Pfaundler, is that the pylorus, which has been found on operation to be thickened and hypertrophied, is simply as found in other normal stomachs postmortem. I have taken up this question in the early part of the paper, and will not repeat any more than to say that some of the cases, which I have recorded as spasm of the pylorus, would fit the Pfaundler theory more completely than the case which I now record.

CASE XII.—*Congenital Hypertrophic Stenosis of the Pylorus.*

The first case of this kind which I shall record was one which came to operation. The patient was a male child, five weeks old, the first child; parental history negative; the weight at birth not known. The mother gave the breast for two weeks. She seemed to have a normal quality of milk. The baby had vomited from birth, vomited after every nursing; the movements were normal, and the vomiting began the first week after birth. It weighed, when first seen, eight and one-quarter pounds. Its general condition was good, physical examination of the organs negative. At first there was no peristalsis, no pylorus was felt. The mother's milk was not so very satisfactory, in that it was not abundant, and was whitish in color. The provisional diagnosis of pyloric stenosis was made. The patient, under observation, was seen to vomit not only everything it had nursed, but seemed to vomit more than it had nursed at a single feeding. In other words, there was residual milk in the stomach. The vomiting occurred about fifteen minutes after feeding and was slightly curdled. The child was taken off the breast and placed on modified milk, and did not improve. It was then placed on two wet nurses, the wet nurse having been changed, with slight improvement only. The bowels were constipated, the movements small, yellowish, and tape-like. It had lost in one month one and one-quarter pounds, having been nine pounds at birth.

When admitted to the hospital, to which it was sent for further observation, the abdomen was tympanitic, and there was visible peristalsis on the abdomen, from left to right, stopping suddenly at the pyloric end of the stomach. The pylorus could be felt two inches below the tip of the ninth costal cartilage, as a round movable mass, the size of a small chestnut. After observation for some days it was found that the peristalsis increased. A wet nurse was procured, but the vomiting continued, the weight having diminished, so that just before operation, which took place a few days after admission, the child had lost three-quarters of a pound, being six and one-quarter pounds. The peristalsis became more disordered

and visible. The child cried after every ingestion of food, just before it vomited. After nursing from the wet nurse, the stomach would erect itself fully three-quarters of an inch upon the surface of the abdomen, as if attempting to drive the food through the pylorus, the child being uneasy all the while, and then vomiting of almost all that the infant had ingested would occur. In view of the fact that the infant was losing ground and looked extremely prostrated, Dr. Lillienthal was asked to see it, and agreed to perform a posterior gastro-enterostomy. After operation, the child continued to vomit on the wet nurse, and was placed on artificial food. The first twenty-four hours after operation nothing was given but water, the child being exceedingly low. He then seemed to come to the surface. He vomited the food and some greenish matter, and finally, when placed on the artificial food, which was retained much better, improved to a remarkable degree and recovered.

I may say, without going into the surgical details of the case, that Dr. Lillienthal, wishing to finish the operation very rapidly, could only partially investigate the condition of affairs in the abdomen. He confirmed the presence of the pylorus, hypertrophied and hard, and fixed to the posterior wall of the abdominal cavity. It is not necessary to go further into the case than to say that after the operation the infant vomited several times a day small quantities of greenish material, feeding having been begun twenty-four hours after operation. After five days of unsuccessful feeding on the breast, the infant not increasing in weight, artificial feeding was again attempted, and after a few accidents, the pulse at one time, having become rapid and feeble, and Dr. Lillienthal having thought the child would be lost, it began to rally, made a very good recovery, and on its discharge from the hospital, it weighed seven pounds and ten ounces, a gain of one pound and six ounces over the lowest weight before operation. The infant then continued to improve under observation at home, and weighed twenty-two and one-half pounds at the age of fifteen months. I have since seen the child at intervals, and it remains perfectly well and has absolutely no gastro-intestinal symptoms referable to the pylorus or operation.

Discussion. This is a case of undoubted congenital hypertrophic stenosis of the pylorus combined with spasm of the pyloric and cardiac end of the stomach, with vomiting to such an extent as to endanger the life of the patient. With the mature judgment of the surgeon and physician the infant was operated upon successfully. It is, of course, impossible to say, in view of the low condition of the child after operation, whether continued attempts at feeding might not have succeeded in this case. It is enough to record, however, that our feeling at the time was that should we wait much longer, we would lose the child, and this, to our conscience, seemed sufficient justification for the operation. The fact that the

child improved after the operation is, of course, no proof that the operation was absolutely indicated; still, it was the only thing that we could possibly think of, if we intended to save the life of the child at the time.

CASE XIII.—*Congenital Hypertrophic Stenosis of the Pylorus; Operation; Death; Autopsy.*

L.S., male infant, aged seven weeks; family history negative. At birth, infant was normal; normal delivery, with no history of previous disease. Nursed at the breast for five weeks, then the breast feeding was omitted and re-commenced. The break in the breast feeding was made by that of artificial feeding, which was continued after the breast was commenced again. Three weeks before admission to the hospital the infant began to vomit; the movements were green curdled; there was a slight cough. It improved for a few days, then became worse and vomited and was constipated, and when the bowels did move, they were greenish. Both ears had been discharging for the six days before admission to the hospital. There was some fever, great loss of weight; the principal complaints were vomiting, constipation, and loss of weight. The physical examination was negative, with the exception that there was a discharge from both ears, the mastoid region was negative; the mouth presented marked stomatitis, tongue coated, the inguinal glands slightly enlarged. The abdomen was lax, tympanitic, nothing palpable, no tenderess, but there was visible gastric peristalsis.

The day after admission we have the following note: Patient vomits within a few minutes after each feeding, peristalsis takes place a few times, then the entire feeding is vomited. No tumor can be felt. There is a sense of resistance in the right hypochondrium in the region of the pylorus. The patient retains nothing on the stomach. The next day a regular feeding was given and the infant studied. Peristalsis was visible before feeding, and became much more marked after feeding. After several waves of peristalsis, the feeding was expelled. The expelled food was at times mingled with a yellow greenish fluid. Immediately after feeding the pylorus could be distinctly felt beneath the liver in the right hypochondrium, between the free border of the ribs and the umbilicus, as a round hard substance.

The next day a test meal was given, and later recovered. The test meal consisted of three-quarters of an ounce of food, and at the end of one-half hour one-half ounce was recovered, a thin whitish fluid showing small milk curds, and moderate amount of mucus; reaction was acid. There was free hydrochloric acid; total acid was 38, combined acids 22; no lactic acid, no blood; microscopical examination negative. The stomach washings were positive to rennet; pepsin positive, but weak on account of dilution, negative as to free hydrochloric; combined acids 6 c.c. per hundred; peptone was present. Two days after this another report of the

gastric contents showed acids 24 c.c. per hundred; free acid absent, positive to rennet, positive to pepsin, peptones present. The vomiting continued, the child losing in weight.

Dr. Lillienthal saw the patient in consultation, and agreed to the performance of an operation for the relief of the pyloric stenosis. The stomach at the operation was found distended and somewhat prolapsed. At the pylorus a smooth mass the size of a walnut was felt, very much like an intussusception; the transverse colon was drawn up over the stomach. A posterior gastro-enterostomy was made, the surgical details of which I will not go into. The temperature before the operation was 103.6°, probably on account of the otitis; unfortunately, after the operation the temperature rose; the region of the left mastoid became swollen, so that relief of this condition seemed imperative. An incision was made, a conservative opening of the antrum was effected; it was filled with pus. The wound was packed in gauze, but the child died twenty-four hours after the mastoid was opened.

Postmortem. Specimen obtained showed the hypertrophic condition of the pylorus and the narrowing of the lumen of the valve. The stomach, however, was not dilated; on the contrary, it seemed to be smaller than normal. The posterior gastro-enterostomy showed no leakage. The infant probably died as a result of the shock of the operation.

Discussion. In this case the unfortunate ear complication, necessitating even a conservative interference, rather militated against the recovery of the child. The good judgment of the surgeon demanded the primary operation, and it seems as though possibly, had the child had a little more resistance, it would have recovered from its malady. It is hard to tell, even in this case, whether temporizing would not have resulted in an ultimately better condition of the child, but the child certainly did badly before the operation, and there seemed to be no other course open to our judgment.

I wish to record a case which shows an interesting phase of this subject, that is, that children exhausted by gastro-intestinal intoxication, starvation, and pain may die suddenly before aid, either through feeding or surgery, can be instituted; that is, while waiting for an improvement or for a decision of therapy, the patient suddenly dies.

CASE XIV.—E. S., female infant, aged seven weeks. The only child; no miscarriages; parental history negative; was nursed for three weeks on the breast, and then the breast was combined with a bottle feeding containing top milk. The present illness began almost immediately after birth; the infant vomited every feeding. Among things which had been tried, the infant was placed on the breast of a wet nurse. There were no movements of any moment, evident constipation. When seen the initial weight could not be

obtained. At seven weeks the infant weighed six pounds; its general condition was very poor, and it was emaciated. To one side of the median line, beneath the liver, and above the umbilicus the contracted pylorus could be distinctly felt. The child vomited at every feeding. A diagnosis was made of congenital hypertrophic stenosis of the pylorus. A test nursing was vomited in the office at the first visit. The infant was temporarily placed on what was considered a better food and a wet nurse advised. The father reported the next day that the child had died in bed.

Discussion. This infant when brought under observation was no worse than many infants I see with similar affections. It was very ill, but there was nothing to warn one, except the extreme weakness of the child, of the sudden demise, and when compared with the following case will be of interest to those who are anxious to know the latest period at which interference of a surgical nature is permissible.

CASE XV.—R. B., male infant, aged eight weeks. First child; seen in consultation with Dr. W. Family history negative. At birth it weighed six pounds and fourteen ounces. It was fed at first with modified percentage mixture, and then was placed on a wet nurse. It was perfectly well and nursed well, and gained in weight until three weeks after birth, when it began to vomit. Previous to this it had always retained its feeding. The vomiting continued. Sometimes three or four feedings a day were vomited, and the others were partially vomited. The infant lost in weight, and after two weeks of experimentation with infant foods and stomach washings the infant was placed on the breast of a wet nurse. It then began to vomit more than before, and the loss of weight was more palpable. When seen first it was in the eighth week of infancy; weighed five pounds and nine ounces, having lost one pound five ounces of its initial weight. Its general condition was exceedingly poor and marantic; the skin hung in folds on the body. The physical examination was negative, but the infant had a double inguinal hernia. It had a mild form of peristalsis, and after a trial nursing there was no vomiting, and I proposed that a new wet nurse be tried. On the new wet nurse the condition was as follows:

There were two, sometimes three, vomitings in twenty-four hours, in which all the food taken into the stomach was rejected, and then the infant would vomit varying quantities of the other nursings. Peristalsis on the abdomen was very apparent, and at times tumultuous, even between the nursings. A small nodular mass, the size of a white bean, could be felt underneath the liver, half way between the free border of the ribs and the umbilicus. The infant seemed to be uneasy, and cried during peristalsis, but nursed with great eagerness. The infant was under observation for five days, and during that time the movements were semifluid, yellow, and to

my mind not sufficient in quantity to show that much food had passed through the pylorus, but only a fractional amount of what it had nursed. At the consultation the advisability of operation was freely discussed, but, on account of the extremely weak condition of the infant, interference was postponed for at least three days, for further observation. The child was given dionin, $\frac{1}{100}$ of a grain, three or four times a day.

The subsequent history of the case is exceedingly interesting. The vomiting continued as well as the peristalsis, but the infant seemed to hold its own. It had gained three ounces under observation, and had fallen back three ounces in the week in which it was observed, leaving its weight five pounds and nine ounces. The infant seemed comfortable, however, and after a few days of additional observation the vomiting became less frequent, and the infant slowly gained. The vomiting finally ceased, the weight increased, but the peristalsis continued for some time, being visible through the very thin abdominal walls. After five weeks the infant had gained ten ounces.

Discussion. This case, after conscientious observation, seemed to be one in which operation was certainly indicated. The operation was only postponed because, to the surgeon, the infant appeared too weak, and its chance of recovery from the operation too small, and still this infant improved without operation, and continues to improve today. The increase in weight is not such as would encourage us to predict the future of the infant; at the same time, no one can convince himself, in face even of an increase in weight of three-quarters of a pound a month, that any surgical interference was warranted. This infant was certainly as weak as the previous infant, which died suddenly, too weak, in fact, to tempt the surgeon to operate, and yet improved in spite of the severity of the symptoms and the gloomy outlook.

DIAGNOSIS. In the diagnosis of these cases we should try as much as possible, from a clinical standpoint, to distingush between the two conditions which I have discussed—those in which there is a spasm of the pylorus without any palpable hypertrophy of the tissues entering into the formation of the valve or the pyloric end of the stomach, and those in which the clinical distinctive features, which have been described, warrant the diagnosis of congenital hypertrophy of the pylorus with spasm of the cardiac and pyloric portions of the stomach, causing a narrowing to a greater or less degree of the valve.

First, in simple spasm there is the persistent and full vomiting; the retentive vomiting present in the cases of congenital hypertrophic stenosis; there is also the steady emaciation and the constipation. There may be no peristalsis, and the pylorus, if palpable at all, is only felt as a small contracted nodule, not as hard or large as in the cases of hypertrophic stenosis. There are

also, once or twice daily, stools which contain a certain amount of milk feces.

In hypertrophic stenosis all the symptoms mentioned are present to an aggravated extent, but in addition there is marked and disordered peristalsis visible on the abdominal surface. This peristalsis is generally in one direction, toward the pylorus, which in a great number of cases is distinctly papable. The constipation is also more inordinate, and the stools show little or no milk feces, only bile-stained mucus.

I have endeavored to distinguish these two sets of cases, of spasm and of congenital hypertrophy of the pylorus, with broad, distinguishing characteristics, and not by indefinite academic lines. The clinical study of the cases will place them in either class.

Congenital conditions, such as real growths of the pylorus or atresia of the pylorus, are exceedingly rare, and can scarcely be brought into consideration in connection with conditions which are considered in this paper. The symptoms in congenital atresia and growths which completely obstruct the pylorus must come on immediately after birth, and are rapidly fatal, unlike the conditions in which the symptoms appear some time after birth. Congenital stenosis of the jejunum or duodenum may be confounded with that of stenosis of the pylorus, if the congenital atresia of the gut is situated high and near the pylorus. There may then be a series of symptoms on the part of the stomach indistinguishable from those of pyloric obstruction. Pure pyloric spasm, I feel, may well occur and does occur with very slight hypertrophy of the pylorus, giving rise to only a limited form of stenosis. Ibrahim doubts the existence of pure pyloric spasm, but I have tried to show that it does occur, and this also in quite a percentage of cases; more especially is this so in those cases of persistent vomiting in which there is sudden or gradual cessation of symptoms upon the inauguration of correct diet and feeding. I think, in considering the question as to whether a spasm or severe form of stenosis is present, one of the most useful clinical guides is the amount and quantity of the stools. If in a given case the stools consist mostly of bile-stained mucus and very little fecal matter, in spite of the ingestion of an ideal food, such as breast milk, we are driven to the conclusion of the presence not only of spasm of the pylorus, but also of narrowing and stenosis of high degree. If, in spite of vomiting at every feeding, peristalsis and even a palpably contracted gut in the region of the pylorus, there is one or two stools daily containing some milk feces, we must feel, as in certain of my cases which at times appeared hopeless, that the stenosis at the pylorus is not of high degree, and that the spasm relaxes at times and allows of a certain amount of food to pass and nourish the patient. It is in most of these cases that we can feel that the ultimate out-

come will be favorable, no matter how exasperating present symptoms appear to be.

PROGNOSIS. The ultimate fate of these cases is extremely interesting in view of the recent contention from some quarters that as soon as the diagnosis of hypertrophic congenital stenosis is made the surgeon must interfere in behalf of the infant. I have tried to show that a large number of cases are really spasm cases, and will eventually recover on internal therapy. Persistent trial of feeding, the most diverse, will eventually result in overcoming the condition.

In one of my cases, a food was finally successful which is known to be an entirely unsuitable one in feeding infants over long periods of time. Why does the spasm relax in these cases, or cease after having continued for months? I have only one theory to offer, and it is that after a time, as in other nervous affections, there is a nerve exhaustion, the system becomes immune to the intoxication, or the intoxicating substance is excreted more rapidly and its action on the mucous membrane and nerve filaments of the stomach becomes nil and inefficient, the nerve tension exhausts itself, the spasm relaxes, and then any food that is digestible and assimilable will be effective in bridging the crsis. Thus, a food that at first would have been unsuccessful will in the relaxed period succeed.

As to the ultimate prognosis of true hypertrophic stenosis, my feeling is that there can be no general statement that will fit all cases. Most cases, I am certain, may be bridged over their crises by persistent attempts at feeding. It would also appear, from at least one of the cases here recorded, that ultimate recovery, even in the face of extreme exhaustion, is not impossible without operation, and we cannot but feel that most cases will escape the surgeon and only the rare cases reach the operative phase. This is only one fact which needs discussion in certain cases in which operation was refused and in which vomiting and other symptoms have subsided and the patients made an apparent recovery. This fact has not been mentioned in any of the many monographs on this subject.

Cessation of vomiting and all distressing symptoms does not constitute a complete recovery; the infant must also increase in weight and develop in a normal manner. I have lately met an illustrative case which in early infancy presented all the symptoms of hypertrophic stenosis of the pylorus and in which the symptoms persisted until the sixth month of life. They then ceased. The infant not only did not gain normally in weight, but developed the most aggravated symptoms of malnutrition. A well-known pediatrist had advised operation; but on reflection this advice was rejected by the parents. Today the child is a rachitic dwarf, aged two years, weighing twenty-one and three-quarters pounds, with a very marked lordosis of the lumbar spine, and a height of only 61 cm. This condition, fortunately extremely rare, is a direct result of lack of nutrition dependent on the pyloric condition in

infancy. Today this child is able to partake of all kinds of food, has no sign referable to the pylorus, but is physically a human caricature. This is an exceptional ending, for most of the cases I have described as recovering have done well, and in the end approached the normal in weight and general appearances. There is another contingency which is striking in those cases which have recovered without operation. That is, in after childhood there remains an indication of pyloric contracture and spasm. A case I saw recently was that of a male child of very good development, who during infancy showed symptoms of spasm referable to the pylorus. The child was brought to me because, whenever solid food was partaken by him he would complain of gastric pain and then vomit the solid particles. Such a case might fit in the class of cases occurring in later life and described by Landerer.

TREATMENT. I shall consider for the sake of conciseness: (*a*) Feeding; (*b*) mechanical means of therapy; (*c*) drugs; and (*d*) operative means.

Feeding. In a given case of hypertrophic stenosis or of congenital spasm the feeding is undoubtedly by far the most important element in the treatment. Breast feeding is the ideal method of feeding these cases, but not every breast will be found adapted to the infant. The breast is given at long intervals and short nursings. Many infants who have not improved on a given breast, or to whom a breast is not available, will be tided over their illness by some of the many and diverse forms of substitutes for the breast at command of the physician. I do not think any artificial food is ideal, and no one is a panacea in this condition. Some insist that the food contain a minimal fat, and I have shown that many cases have recovered on food which all pediatrists agree is the most unsuitable in the long run under ordinary conditions. In other words, though this condition seems in a certain proportion of cases to have been inaugurated by some error in diet, there is no royal road to the feeding. In artificial as in breast feeding the method must presuppose small amounts at each feeding, at long or short intervals, as the case may be.

Mechanical means of therapy include the application of warm cataplasms of flax seed and hops, or dry warmth, stomach washing, and enteroclysis. Stomach washing, I feel, is a useless and in some cases, when the infant is in a weakened condition, an exhausting procedure. Some of the cases which are detailed in this paper had been treated early by stomach washing without avail, and others with a distinct exacerbation of symptoms. All of the cases I have noted have eventually recovered without it. Enemas are useful in the form of enteroclysis of small amounts of normal saline solution to maintain nutrition. They are given several times daily.

Drugs. Heubner advises opiates, others derivatives of opium, in very small amounts to quiet the spasm of the pylorus and adjacent

stomach wall. Heubner uses the tincture. In most of my cases no opiate was resorted to, and in only one was it given, and then only after improvement was well inaugurated and only in exceedingly small doses and at desultory intervals. I have found but temporary benefit from the administration of citrate of sodium, or sodium and pancreatin.

Operative Therapy. An operation such as is proposed for the relief of congenital hypertrophic stenosis of the pylorus presupposes great technical skill on the part of the surgeon, and great dexterity and rapidity. One surgeon may complete the operation of posterior gastro-enterostomy in fifteen minutes, another in forty-five minutes. The difference to the patient can better be imagined than described. Again, a mortality of 50 per cent. (Ibrahim) to 75 per cent. is certainly not an encouraging prospect for the physician to hold up to the anxious parents. We are asked to hand these cases to the surgeon before it is too late. Who will say, and how are we to decide the proper moment when all further attempts to save the patient will be hopeless. I confess, with all my experience with these conditions, I still feel that my chances with nature are as good, if not in some cases better, than with the operative measure.

There is another point which I especially wish to consider, and that is, that the cutting out of a portion of the gut, which results when posterior gastro-enterostomy is performed, is not without harmful influence. The infant who has been recorded in this paper as having been successfully operated upon, did very well up to the eighteenth month of life, and presented no symptoms referable to the operation. He has, however, lately shown an anemia which does not yield to treatment, a rachitis of very marked degree, and late in its development. He presents also respiratory apnœa and, what is most peculiar and especially interesting to the surgeons, symptoms which point to a narrowing of the artificial opening in the stomach. The child is beginning to vomit after the ingestion of solid food; even small pieces of cracker cannot be partaken of without danger of a vomiting spell following immediately or soon after the meal. Meat the little fellow refuses to take, inasmuch as he feels that he will have to reject it. About two years after the operation symptoms of possible stenosis of the artificial opening in the stomach are beginning to develop.

THE THERAPEUTICS OF SELF-REPAIR.[1]

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Numerous facts in biology justify the conception that the living organism represents a mechanism, and is in very many respects comparable to a complex human-made machine. Such a view proved also to be practical and very fruitful in the investigations of biology and medicine, since it encouraged the extensive study of the details of the living mechanism by the well-established methods of physics and chemistry. For therapeutics, however, this view seems to contain an inimical and discordant elemen . Therapeutics means the principles and methods employed in the repair of the disordered living organism. Now, the efficient repair of the mechanism of a human-made machine justly presupposes a familiarity with its normal structures and construction, and also a complete understanding of the nature of the disorder. But what is the status of our understanding of the living machinery and its disorders? Well may we point with pride to the great strides we have recently made in this understanding. But what is the actual extent of our knowledge? It may be freely admitted that it is very small, and is only an infinitesimal fraction of that knowledge which would enable us to comprehend the living machine as a competent engineer understands the human-made machine. And what is more, in the human machine physical and chemical forces may be, and probably are, active, but of their nature the present sciences of physics and chemistry have not even an inkling. Shall we follow the requirements of the human-made machine and wait with our therapeutics until we know all that?

While, thus, no man would be permitted to meddle with some serious disorders of a human-made machine with only a fraction of knowledge of its construction, we find that with the disorders of the mechanism of li'e for thousands of years millions of men meddled with their repair with a knowledge infinitesimally small indeed, even when compared only with the present-day understanding of the processes of life and their disorders. The contrast is still more striking when we consider the differ nce in the results. No man can be in doubt as to the outcome of an ignorant meddling with a human-made machine; it would lead, in the great majority of instances, to its complete destruction. But with the human organism, no such disastrous results followed the innumerable ignorant meddlings with its disorders; at least, such evil results were not

[1] Read at the meeting of the Association of American Physicians, Washington, D. C., May 12 and 13, 1908.

readily recognizable: otherwise medicine would long ago have been wiped out of existence.

The truth of the matter is, that with regard to therapeutics the living animal body ought not and cannot be compared with an artificial machine. With respect to repair, there is a fundamental difference between a human-made machine and a living organism. The disorders of a machine can be repaired only by human hands. No machine has yet been invented which can automatically repair its own disorders. The living organism, however, is well provided with such automatic arrangements for self-repair. This fact has a far-reaching theoretical and practical importance. The fact itself, namely, that the living organism can master, unaided, its own disorders, was recognized very early in the history of medicine. Its true significance, however, was never fully grasped. In olden times, as well as in our modern era, it was utilized only as a weapon to discredit the claims of artificial therapeutics. It was pointed out over and over again that patients get well without any treatment, and if they get well under treatment, it might be that they do so, not because of it, but perhaps even in spite of it. The scientific labors of recent years, however, demonstrated clearly that the knowledge of the body's great ability for self-repair, far from being an inimical element to therapeutics, can be utilized as an element of great strength in support of the human efforts to cure or to hasten the cure of its ills. In the brief time at my disposal I shall devote my attention chiefly to a discussion of the aspect of therapeutics from the point of view of our knowledge of self-repair. I wish, however, to preface my remarks with the following brief statements, which, although self-evident, are nevertheless not superfluous.

Science and practice of medicine exist for the benefit of the sick, and therapeutics is the most important part of medicine. To be content with the mastery of pathological anatomy and diagnosis, and to have a contempt for treatment, is a moral and intellectual anomaly. This is, as suggested above, a self-evident truth, which, however, is frequently sinned against, and the sinners are to be found more among the leading clinicians than among the average practitioners. Furthermore, the fact that the living organism is provided with means of self-repair does not relieve us of the duty to strive to discover and procure also artificial remedies. Evidently, Nature's store of means of repair is insufficient; otherwise there would be no suffering and no premature deaths.

Two methods of treatment competed in the past in the domain of internal medicine: the rational and the empirical. I shall not discuss them further than to say that the rational method possesses mainly negative virtues and leads essentially to inactivity. The empirical method, which satisfies its followers by the activity to which it inspires, repelled the active minds in the profession by the vagueness of its inceptions, by the tediousness of its procedures, and

above all by the unreliableness of its results. In this despair the active minds found consolation in the study of the normal and abnormal structures and constructions of the human mechanism, leaving the question of treatment entirely out of consideration; thus medicine was threatened with the loss of its essential object. Since it was generally admitted that many ailments and diseases terminate in recovery, it was evident that the animal organism must possess means and methods by which it accomplishes these recoveries. Here, then, a competent school exists where therapy could be learned legitimately and efficiently. With this object in mind, the methods of self-repair of the animal body became the object of profound scientific studies, and immunity became the third method of therapeutics. This statement deserves to be especially emphasized. The study of immunity means the study of the natural therapeutics of the body with a view of employing it also artificially and, when possible, improve upon it. From this point of view immunity is the most rational and most promising method of therapeutics. I am afraid that this aspect of the studies of immunity is not yet appreciated even by some leading pharmacologists.

The difficulty with which these new studies were confronted was to discover and discern during the process of a disease the phenomena which were manifestations of repair. All recognizable phenomena had already their assigned places; they were pathological if they were not physiological; all deviations from the normal were parts of the disease. It was a good fortune that the antitoxins were discovered; here was something which physiology could not claim as its own, and pathology had not yet laid hands upon. Here was something which the body in its effort of defence and repair evidently manufactured anew. And I may add that here the new therapeutics was quick enough to learn to improve upon the body's method: to provide with passive immunity when active immunity failed. The new field is rapidly growing larger. Besides the discoveries of new substances and processes, many old phenomena are now reclaimed from pathology in behalf of the domain of self-repair. To quote a few trivial instances: pains of joints or muscles are looked upon as a means of insuring immobility of the diseased parts; cough is a means to remove irritating, infecting, or obstructing agencies from the air passages; the production of mucus in gastric ulcer is a means of protection for the denuded tissues from digestion by the gastric juice; vomiting or diarrhœa is a means of relieving the gastro-intestinal canal from corroding or intoxicating substances.

The present conception of the pathology of inflammation is an instructive and interesting illustration of the point under discussion. Tumor, rubor, calor, and dolor were the old insignia of inflammation. The modern pathological studies distinguish local and general symptoms. The local symptoms consist of hyperemia with

stasis, emigration of leukocytes, exudation of lymph, increase of glandular secretion, proliferation of tissue cells, and formation of granulation tissue. Of the general symptoms, we should mention only fever and leukocytosis. All of these symptoms were considered as manifestations of the disease. The present tendency in pathology is to consider every symptom as a manifestation of the efforts of the organism for defence and self-repair. The profusion of blood and lymph, with their natural bacteriolytic, antitoxic, and antifermenative properties; the leukocytes, with their phagocytic and digesting activities, and the lymphoid and granulation tissues, with their mechanical and vital powers, attempt to destroy and remove, or at least to wall off, the primary causes of the disease, to neutralize the secondary intoxications, to separate, dissolve, and remove necrotic tissue, and to start regenerative processes. Of the general effects, leukocytosis is now generally acknowledged as being a strong means of defence. Fever, so far as the elevation of temperature is conconcerned, was considered at various epochs of the history of medicine as a sign of the body asserting itself against the disease. It is now assumed by some writers that it assists in some way or other in the termination of the infection. In short, all the signs of inflammation are now considered as factors in self-repair.

It is interesting to observe how, on the basis of this newest conception, some of the very same signs which were previously considered as essential manifestations of the disease are now artificially produced for therapeutic purposes. There is to be mentioned, in the first place, the production of venous hyperemia in the treatment of various acute and chronic diseases of the body, the so-called Bier's method. The results seem to be excellent indeed. Here we see a brilliant surgeon developing a practical medical method of treatment on the very basis we have been discussing here: the therapeutics of self-repair. I call it a medical method, because it requires no knife and no anesthetics, and should be learned and employed by every practitioner.

The various attempts to influence favorably the course of infectious diseases by the artificial production of leukocytosis is another venture to utilize therapeutically one of the methods of defence and self-repair employed by the body against the cause of inflammation. As examples, may be mentioned Landererer's method of treating tuberculosis by intravenous injections of hetol; Löwy and Richter's experiments upon the production of leukocytosis by pilocarpin, spermin, and albumose-like bodies; Mikulicz's attempts to increase the peritoneal resistance to postoperative infections by the injections of nucleinic acid, as a leukotactic substance.

Fever is another means of repair which is now being tried by various investigators as a curative agent. Here may be mentioned Buchner's experiments to cure anthrax by fever brought on by injections of sterilized emulsions of Friedlander's pneumobacillus;

Dehio's method of treatment of lupus by fever produced by albumoses; and Bier's attempts to produce curative fever by the intravenous injection of alien blood.

Finally, we have to mention the recent interesting experiments of Opie, in which tuberculosis of the pleura and lungs of dogs was cured or greatly mitigated by the intrapleural injection of sterile pus obtained from another animal. It is again the therapeutic employment of a phenomenon of self-repair occurring in the local process of inflammation, namely, the phagocytic and proteolytic cell activity of the emigrating leukocytes.

I have dwelt upon the phenomena of inflammation at greater length because they illustrate the modern tendency in pathology to look upon some of the pathological manifestations as Nature's efforts for self-repair, and at the same time show the tendencies of our day to learn in therapeutics from the methods employed in Nature.

With regard to the latter, I wish, however, to append here a f w qualifying remarks. In the first place, learning the methods of Nature should not imply that we have to follow them blindly; on the contrary, we ought to try to improve upon them. Nature's methods of repair often lead to disastrous results; I need only refer to strictures of canals following the natural repairs; or to the formation of abscesses in important organs without the possibility of a spontaneous innocent outlet. In studying self-repair, we need only to get the hint as to Nature's methods; but he is a bad pupil who does not try to do better than the master.

Furthermore, even if we acknowledge that certain phenomena of disease are Nature's methods of repair, it does not follow that we are bound to tolerate their continuation in all cases. In some instances we are at liberty to assume that the method is wrong and that the treatment might be even worse than the disease. For instance, even admitting that pain in articular rheumatism is a part of repair by thus insuring the immobility of the joint, we need not and should not accept this method of treatment. Immobility can be better attained by a splint, and we should, therefore, relieve the pain by an opiate or by a salicylate. Or, to take another instance, even if it be true that fever may be beneficial in some cases, it is wrong to permit its excess when it obviously makes the patient feel miserable. Besides, we should not be orthodox, and should not sacrifice the patient's comforts to mere theories. I am not willing to accept Nature's advice to treat the disease by fever to the great discomfort of my patient, as I am not willing to treat the fever with the cold bath, when, to use Osler's description, the patient is in dread of it, and pitifully begs to be left in the warm bed. We ought also to bear in mind that our duties differ from the self-imposed task of Nature. Nature strives only to avoid death; the physician has an additional duty, and that is to make life bearable. Furthermore, we should not lose sight of the fact that relieving from suffering may mean much more than symptomatic treatment; in some cases it may mean saving life. I

need only refer to one well-known fact, that general or local anesthesia may save life by avoiding shock. The recent statements that the fatal effect of anaphylaxis can be obviated by ether or chloral anesthesia contain, to my mind, a very significant principle, which should not be lost sight of in therapeutics.

Without dwelling on the subject further, I wish to emphasize my view, that phenomena of disease, when they cause serious discomfort, ought to be treated, even if they belong to the group of self-repair, provided, of course, the treatment is not endangering life.

Returning to the methods of repair of the animal body, we may say in general that the living organism responds with a reaction to any change in its normal condition; the reaction may be local or general, or both, and it may be in the nature of an increase of the normal forces, or a change in the distribution of forces; or a call upon the reserve or the latent forces; or, finally, the reaction may consist in the creation of entirely new forces.

The phenomena of inflammation illustrate the several varieties of the first-mentioned reactions. New creations are the antitoxins, bacteriolysins, anti-endotoxins, agglutinins, precipitins, and coagulins. These phenomena have been recognized only within recent years, and it is entirely premature to assume that the above list represents all the new substances which the organism is capable of producing in its struggle against disease.

The therapeutics which is based upon this new lesson from Nature has already attained great success. I need not speak of diphtheria antitoxin; its usefulness is beyond doubt. Last year's experimentation established the practical efficiency also of an anti-endotoxin. I mean the serum for meningitis. It is my impression that its practical efficiency is as good as that of the diphtheria antitoxin, if not better. The other methods of treatment by passive and active immunizations I need not dwell upon, as they will be treated by another speaker.

The enumerated instances will suffice to prove my contention that the fact of spontaneous recovery of the organism from disease can be, and has already been, utilized with great profit in favor of scientific and practical therapeutics.

The fact of spontaneous recovery has frequently, as stated above, been utilized as an argument against the use of any artificial treatment; and if the usefulness of any treatment was admitted, it was that of physiological therapeutics; that is, to improve during disease the conditions which the body requires during health. In the foregoing we have learned that there are methods of treatment which, in contradistinction to the term physiological therapeutics, may be designated as pathological therapeutics; that is, the employment of methods of treatment along the lines which the organism institutes during disease in its efforts of defence and self-repair. Pathological therapeutics promises to be the most efficient method of studying therapy.

SAFEGUARDS OF THE HEART-BEAT.

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The student of disease loses nothing in accuracy and may gain much in comprehension by pausing now and then to contemplate the principles involved in phenomena that are trite and familiar. Thus, to preserve the equilibrium of the upright body there is a single physical condition which is all-essential, namely, that a vertical line dropped from the centre of gravity of the body shall pierce the area circumscribing the feet. Let the frame be bent to any extent in one direction, equilibrium may still be preserved by a definite mechanical displacement in the opposite. Whether the area of support be large or small, the distortion of the figure great or little, the body will still stand provided its centre of gravity remain within the area of support.

What is this but *compensation*, which secures the realization of a purpose by maintaining the balance of opposing forces? Turning to the almost infinitely complex system of units of matter and energy whose equilibrium we know as Life, we always find that a departure from a mean in one direction is accompanied by a corresponding deviation in another; so that the terms "health" and "disease" find their best definition, not according to some specific adjustment of structure and function, but according to the efficacy of the balancing of ever-shifting forces by which life is maintained. We are wont to think of compensation in vital activity as some unusual adjustment to meet a critical functional failure; whereas, probably every physiological feat accomplished manifests the balancing of equal and opposite forces. The principle here involved is perhaps the main factor in biogenesis, but, nevertheless, students are prone, especially on the clinical side, to neglect its application.

While we must ascribe most adjustments of living structures to environment to slowly acting natural selection, the history of a single individual may prove the intimate dependence of growth upon physical conditions. Thus, Babak[1] has shown that the external gills of amphibian larvæ grow luxuriantly when the animals are kept in water poor in oxygen, but attain progressively smaller dimensions when the oxygen pressure is increased; that is, the oxygen-absorbing organs wax or wane in volume in order that the tissues they supply may receive the needed complement, no more, no less, of the vitalizing element. To the same physiological category belongs the familiar fact that the pupil of the eye dilates in the dark and contracts in the light, whereby the sensory cells of vision escape overstimulation and appreciate feeble illumination.

[1] Ref. in Biophysikal. Centralbl., December, 1907, 191.

Sherrington has demonstrated that even a simple motor impulse to a skeletal muscle involves a simultaneous inhibition of the tonus of the antagonistic muscle group. The mechanisms of the body are probably all radically imperfect when measured by the demands we make of artificial instruments. Yet it will doubtless be found in every case that each dereliction from mechanical perfection is so balanced by compensations that the range of usefulness of the organ to the body is thereby positively enhanced. For example, the eyeball is commonly considered to have its analogue in the photographer's camera. But its imperfections as such an instrument would make it useless in the arts for the simple reason that its whole surface is permeable to light. But light that enters the eye by any part other than the pupil must have penetrated many bloodvessels and become red in color. Under ordinary conditions of vision, then, the retina is bathed in diffuse reddish light, except for a spot in the centre upon which falls the white light which has entered through the pupil. According to the laws of physiological contrast, the sensitiveness of the retina in this area must be kept ever keen for the complementary color to that which surrounds the central spot, and that color is green. As I have already pointed out,[2] the psychic effects of the verdure of Nature, the uncloying joy of its greenness, find herein a physiological basis. Moreover, it has also been demonstrated that acuity of vision, which measures the optical fitness of the eye, is preëminently keen when objects are viewed in a light of greenish tinge. When, in a sensory apparatus, we find that the most hopeless physical defects are turned into the finest uses of the instrument, we may grant, without prolonging this line of analogy, that there is overwhelming *a priori* probability that every vital organ must be abundantly provided with efficient means of circumventing disaster from the overstrains which the exigencies of living necessarily more or less habitually entail.

The physiologist has no compunction of doubt in describing the heart as essentially a force pump, the continuity of whose current implies the efficient closure of the auriculoventricular valves. It is easy to demonstrate in the excised, normal heart that the perfection of valve action should leave nothing to be desired. Indeed, on forcing water into a human left ventricle, having somewhat fatty walls, through a tube tied in the aortic orifice, I have caused the heart wall to burst while the mitral valve refused to leak. When one considers the limited range of dilatability of the cardiac cavities, the extraordinary variability of the inflowing blood current, the readiness with which the heart may be overstrained by increase of the diastolic blood pressure, and the irretrievable disaster entailed by prolonged asystole, it would seem on *a priori* grounds that there must be some

[2] On the Physiological Effects of Light which Enters the Eye through the Sclerotic Coat, Jour. Phys., 1884, 132.

adaptation of the cardiac mechanism, not obvious in its functional structure, by which the perils of these conditions may be averted. This is practically the conception at the basis of the epochal work of T. Wilkinson King,[3] who, in 1837, published "An Essay on the Safety-valve Function on the Right Ventricle of the Human Heart; and on the Gradations of this Function in the Circulation of Warm-blooded Animals." King demonstrated on a large series of mammals that when the right ventricle is distended with water introduced through the pulmonary artery, there is a reflux of fluid into the right auricle. This insufficiency of the tricuspid valve is brought about by stretching, under pressure, of the yielding wall of the right ventricle to such an extent that the valve flaps are prevented from coming into approximation. Clinicians are daily in contact with examples of "relative valvular insufficiency" occurring in patients having dilated hearts. But when, according to clinical symptoms usually relied upon, there is demonstrable leakage of the tricuspid valve, it seems a reproach of Nature to describe this as a safety mechanism, for the patient is already in desperate straits. From clinical observation as well as theoretical considerations I have long held the view that overdistention of the ventricles is guarded against by compensatory insufficiency of the auriculoventricular valves which is so frequent as to be properly classed among physiological phenomena.

The physical conditions of the cardiac circulation are such as presumably to permit extensive regurgitation from either side of the heart without notable functional disturbance. Each auricle receives its blood stream under a low head of pressure from a reservoir having a capacity many times as great as the chamber which it is to supply. The walls of these reservoirs, the pulmonary veins and capillaries for the left auricle, and the venæ cavæ and their branches for the right auricle, are peculiarly extensible. These channels are capable of accommodating, probably without very notable increase of intravenous pressure, an amount of blood much in excess of that usually contained therein. The term "reservoir" which has of late frequently been used to designate these vascular territories is really descriptive of their function. It is to be expected that any backflow from the heart which does not so raise the venous pressure as to impede the circulation in the vital organs, may be tolerated without producing physiological disturbance. It may, perhaps, also be assumed that at the beginning of a given systole, the regurgitation of an amount of blood which is very small in proportion to the capacity of the venous reservoirs, might save the heart from dangerous overstrain; and toward the end of systole such backflow could prevent accumulation of residual blood. It is obvious that these considerations present a theoretical means of protecting the heart against overstrain during a temporary excess

[3] Guy's Hospital Reports, 1837, ii.

of inflow without in any way interfering with the work of other organs; in other words, such regurgitation from the ventricles would represent a true "safety-valve" action of the auriculoventricular leaflets. As will be pointed out later, it is to be expected that such valvular insufficiency would altogether escape ordinary observation because the amount of blood thus injected into the auricles and veins is not sufficient sensibly to distend them. But when the backflow through the auriculoventricular valves persists, the excess of blood entering the heart over that leaving it is stored up in the auricles and veins, and leads to a progressive increase of venous pressure. In such an extremity the veins are engorged and may pulsate visibly like arteries with contraction of the ventricle, because the elasticity of their walls has been pushed to the limit. The heart is now permanently dilated; the auricles may be fixed in paralytic distention, and the atrial rings are so stretched that their valves are relatively insufficient. Nevertheless, the disastrous march toward venous plethora and arterial ischemia may be checked at any point whenever the forces of compensation are able to equalize the rate of inflow and outflow of the heart. Wonderful as it seems, the physiological demands of the body may be satisfied indefinitely by a circulation maintained by a pump with radically defective valves.

Clinical observers, for the most part, appear to regard the occurrence of tricuspid insufficiency as a late link in the chain leading to heart failure—an event manifested by engorgement and pulsation of the central veins, enlargement and pulsation of the liver, and a well-marked systolic murmur audible over the precordium with maximum intensity in the tricuspid area. Gibson, however, is quoted by Babcock[4] as thus characterizing tricuspid regurgitation: "It is incomparably the most common of valvular lesions, and the reason this fact is not brought out in statistics upon the relative frequency of valve defects is to be found in the circumstance that incompetence of the tricuspid valve does not in itself seriously impair the general course of the circulation, and it is therefore often found among those, who, although under treatment for various affections, have no cardiac symptoms. It accordingly escapes observation unless especially sought for." Babcock deprecates this statement of Gibson's which is, however, quoted here because it expresses exactly the conclusions to which my own clinical studies have led. The class of cases which affords the evidence for this belief is one in which there are usually no subjective symptoms and few of the classic signs of cardiac disorder. In fact the subjects have not heart disease, as a rule, but only the functional expression of cardiac overstrain. There is often even no complaint of shortness of breath on exertion. A systolic murmur, usually faint and soft, with much the quality of the normal inspiratory sound in the lungs, is often heard

[4] Diseases of the Heart and Arterial System, 1903, 343.

with greatest intensity along the lower left border of the sternum. But the auscultatory signs in this region are too various and uncertain in origin to permit of diagnosis by the murmur alone. Careful outlining of the heart by percussion usually shows the area of dulness to be somewhat increased to the right, but this sign is by no means distinctive of valvular insufficiency.

In an admirable series of experiments, Roy and Adami[5] long ago studied the time relations of movements in various parts of the dog's heart. They compared the movements of the cardiac wall with those of the papillary muscles by means of tracings obtained from levers attached to hooks, one of which was plunged into the surface of the ventricle and the other inserted through the edge of a mitral valve.

Their records show that at the beginning of ventricular systole, during the period of "rising tension," the edge of the valve is forced somewhat into the cavity of the auricle, but that the terminal act of ventricular systole is marked by a sharp downward jerk of the valve as the wave of contraction invades the papillary muscles; and it is, according to them, this sudden increase of tension which throws open the aortic valves. Forewarned of the danger of depending upon *a priori* arguments, we may still assume provisionally that, were the wave of contraction to fail to involve the papillary muscles, these bands would be stretched like elastic cords, and, as the ventricular parietes approximate in systole, a leakage through the up-pushed valve should be expected. It is evident that the period in the cardiac cycle at which such regurgitation would occur might either cover the whole period of ventricular contraction, or be limited to the early phase thereof in case the contraction wave progressed with unusual slowness, or it might be restricted to the end of the ventricular outflow in case the contractile tonus first abandons the papillary muscles. Tracings of the venous pulse suggestive of the latter condition are obtained with great frequency. My conception of functional contraction failure of the papillary muscles was suggested by the analogy of results obtained many years ago in experiments upon the skeletal muscles. When a lever is laid over each end of a frog's muscle, which is fixed at one extremity and rests upon a horizontal support, the contraction wave, started at one end of the muscle by a submaximal stimulus, is found to diminish progressively in height as it traverses the fibers. As the stimulus is weakened the wave involves less and less of the length of the fibers. When a third lever, in connection with the free end of the muscle, registers its shortening, it is found that the length of the latent period of contraction varies inversely with the strength of the stimulus; that is, the greater the length of the inert elastic tissue that is interposed between the contracting moiety of the muscle and the lever, the more time it requires

[5] Heart-beat and Pulse Wave. Practitioner, 1890.

to bring the inactive part of the muscle into tension sufficient to lift the weighted lever.

The analogy between these results and the facts of cardiac activity is not far to seek. Physiological experiment on the excised heart muscle shows not only variability in the rate of progress of the contraction wave, but, at least in cold-blooded animals, an extraordinary tendency for the contraction, started at one end of a bundle of fibers, to die out before it reaches the other. The clinical phenomenon of "heart block" is sufficient evidence that somewhere in the contractile chain a conducting link is defective.

Students of the heart-beat have generally been impressed with a something like specificity in the functions of the papillary muscles. In another place[6] certain evidences in this direction are thus presented: "J. G. Adami,[7] summarizing the results of experiments made by Roy and himself on the dog's heart, speaks of the contraction of the papillary muscles as 'virtually independent' of that of the ventricular wall. 'The more we have studied the tracings obtained under various conditions, the more we have been led to conclude that the moment when they begin to contract is not primarily dependent upon the moment of commencing ventricular contraction. We find, for example, that an overdose of liquor strychninæ may lead to complete asynchronism between these two components of the ventricular action; or, again, there may be a ventricular systole unaccompanied by papillary contraction, or vice versa. Fenwick and Overend[8] studied the contractile movements of the papillary muscles and apex of the excised heart of the rabbit. They found that the papillaries began their contraction later than the apex, and they state: 'We never observed a difference as regards the commencement of the curves of less than one-twentieth of a second. The papillaries, however, speedily lag more and more behind the ventricles until they cease altogether.'"

Haycraft and Paterson,[9] in repeating these experiments, find that in the fresh heart the papillary muscles contract simultaneously with the ventricle. Later the papillary muscles may begin to contract before or after the ventricular wall. Fenwick and Overend continue: "It is well known that these muscles [the papillaries] are particularly prone to all forms of structural degeneration, and that their condition constitutes the most delicate index of the general state of cardiac nutrition. In fact they might be termed the 'cardiac extremities,' since, from their single attachments to the ventricular walls, they must necessarily derive their entire blood supply from the nutritive

[6] On the Clinical Relations of the Papillary Muscles of the Heart. Philadelphia Monthly Med. Jour., September, 1899.

[7] On the Action of the Papillary Muscles of the Heart. Proc. Cambridge Philosoph. Soc., vii, part II.

[8] Report on the Contraction of the Papillary Muscles, and its Relation to the Production of Certain Abnormal Cardiac Sounds. Brit. Med. Jour., 1891, i, 1117.

[9] The Time of Contraction of the Papillary Muscles. Jour. Phys., 1896, xix, 262.

vessels which enter at their bases. In short, their circulation must be of a terminal character. It is, therefore, obvious that the *musculi papillares* are at the outset even structurally predisposed to exhaustion and degeneration, and any condition which tends to lower the nutrition of the heart as a whole must affect them with a magnified degree of intensity."

In reflecting on the feasibility of obtaining experimental evidence of functional tricuspid insufficiency, it seemed probable that clinical studies of the jugular pulse might furnish the desired facts. From the very nature of the conditions assumed to underlie such circulatory failure, it is manifest that the clinician and not the physiologist can find ready at hand appropriate material for observation. At the very outset of these studies I was astonished at the unexpected wealth of material presented in the persons of patients whose hearts would pass as perfect to ordinary clinical examination. One easily becomes convinced that the vascular movements of the neck are concerned much more prominently with changes in the veins than in the arteries. The observations can usually only be made while the subject is in the recumbent position. When this posture is assumed, a rapid, more or less, dicrotic undulation of the skin above the inner ends of the clavicles is common and signifies variations in the volume or tension of the great vessels beneath. But, to one who has not definitely investigated the subject, it will cause surprise with what frequency the external jugular veins themselves become prominent and actively pulsate.

With a stethoscope applied to the heart, it is plain in this class of "normal" subjects that the swelling of the external jugular vein usually begins just before, and reaches its acme during the first sound of the heart; it is common, perhaps usual, for the blood wave to distend the vein with a dicrotic motion. When a jugular vein is clamped by finger pressure high in the neck, it drains more or less completely. A pulsatile filling of the vein under these conditions seems to postulate a true systolic regurgitation from the auricle or ventricle rather than a mere rhythmic interruption of the stream which normally traverses the vein. But more commonly it occurs that the swelling and pulsation of an external jugular vein completely disappear when the vessel is occluded above; therefore, the important conclusion is deduced that *venous pulsation does not necessarily predicate the translation of blood mass from below, but only that of a mechanical impact.* Not infrequently, with each heart-beat the neck swells laterally along the course of the carotids. That this slow wave is due to engorgement of the internal jugular trunks and not to arterial pulsation seems proved by the fact that it ceases if the fingers are pressed gently down behind the inner end of the clavicle; at the same time the external jugular vein on the same side becomes turgid and its pulsation ceases. The pressure has simply blocked the communication between the jugular branches and the heart without interfering with the

carotid blood flow, which still may transmit its abrupt pulse wave to the tissues.

Graphic tracings may be made of these various pulsations. When the transmitting tambour is placed over the carotid artery under the angle of the jaw, the tracing obtained closely resembles the sphygmogram of the radial pulse. When the tambour is applied to the area of undulation above the clavicle, the record may still have the characters of the arterial sphygmogram or it may take on quite a new form, that of the venous pulse.

According to Mackenzie[10] the occurrence of a venous pulse always indicates a certain degree of cardiac insufficiency and "limitation of the field of physiological response," recovery from which depends upon the degree of overstrain to which the myocardium has been subjected. In certain cases it can be noticed that a typical venous pulse tends to assume the arterial form after the subject has taken a few deep breaths. With the registering apparatus usually employed, the venous pulse is found to be made up of a group of three characteristic waves (*v*, *a*, *c*, Fig. 1), a special discussion of which is not intended here.

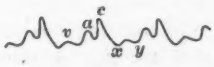

FIG. 1.—Typical venous pulse. The depression *x* marks the extreme relaxation of the auricle, and *y* the corresponding state of the ventricle.

The *a* wave is admitted to be synchronous with the contraction of the auricle and to be caused by it. The *c* wave is synchronous with contraction of the ventricle, though the cause of its formation is in dispute. When the levers registering the venous and the carotid pulse are placed in the same vertical line on a (travelling) smoked paper, it is found that the *c* wave begins about the period of outflow into the aorta, as marked by the ascent of the carotid tracing. The *v* wave, in the class of cases here under discussion, usually begins about the end of the period of outflow from the heart, marked on the carotid tracing by the dicrotic notch. The origin of the *v* wave is ascribed by Mackenzie to the rising pressure within the auricle due to the constant inflow of blood during the ventricular systole. The sudden decline of the *v* wave is dependent upon the emptying of the auricle into the distending ventricle, after completion of ventricular systole. Among patients presenting themselves for examination on account of a wide range of functional disorders, I have been struck with the uniformity with which evidences of cardiac insufficiency could be distinguished, based both upon the nature of the symptoms and the

[10] The Study of the Pulse, 1902.

character of the venous pulse. I have been led to believe that functional insufficiency of the tricuspid valve is of frequent occurrence in such cases, and that it occurs without extraordinary dilatation of the heart. Objective evidence of such regurgitation is furnished by certain forms of the venous pulse. Thus, in Fig. 2, it is seen that the *v* wave has a double crest, or, rather, the wave *v* which begins just

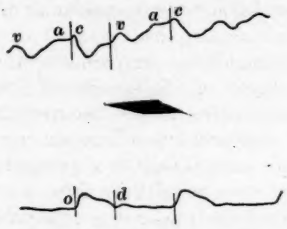

FIG. 2.—From an anemic girl aged fourteen years. The upper tracing is that of the right internal jugular pulse, showing a double crest to the *v* wave. The lower tracing is from the carotid artery; *o* is the beginning of ventricular outflow, and *d*, at the dicrotic notch, marks the cessation of outflow. Events on the same vertical line in the two tracings are simultaneous.

at the moment of closure of the aortic valves, as determined by the dicrotic notch in the lower tracing, is immediately preceded by a wave which is completed during the last moments of ventricular outflow. At this time the auriculoventricular valves should be firmly closed and there is no apparent reason why the gradual increase of auricular diastolic pressure, as marked by the *v* wave, should be interrupted.

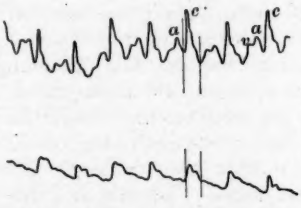

FIG. 3.—Tracings of the right internal jugular and left carotid pulses from an athletic boy aged eighteen years, who had overstrained his heart with exercise. Note the undulations on the descending limb of the *c* wave, which can hardly be due to instrumental defects.

But if we may assume that toward the end of ventricular systole the papillary muscles relax, it is obvious that the tricuspid valves might be lifted by the intraventricular pressure and allow the regurgitation of a certain amount of blood which might cause the first elevation of the wave *v*. It is obvious that the earlier in the systole of the ventricle the assumed papillary relaxation occurs, the earlier is the projected wave depicted upon the tracing; thus, in Fig. 3, the

notch on the descending limb of the *c* wave apparently illustrates such a condition. Marked insufficiency of the papillary muscles occurring early in ventricular systole might readily so reduce resistance to regurgitation that the arterial outflow would cease and the semilunar valves be closed prematurely.

The well-known variations in the height and extent of the *v* wave which depict the progress of early cardiac insufficiency are readily explained by conditions like these. These graphic symbols have their symptomatic counterpart, and, in a rather limited experience, I have been gratified with the therapeutic results of treatment directed to the conservation of cardiac energy in such cases. Mackenzie exhibits many tracings which represent the double apex of the *v* wave. In his explanation of the double crested *v* wave, he attributes the portion preceding the notch, which coincides with the terminal period of arterial outflow, to the same accumulation of blood within the auricle, which, according to him, causes the usual *v* wave, while he assumes that the portion of the wave following the notch to be due to tricuspid regurgitation resulting from the resistance to backflow into the auricle suddenly falling below that offered to outflow through the pulmonary artery. Such an explanation would involve a shortening of the period of outflow, for which there seems no evidence, and, according to pulse tracings, an extreme prolongation of the period of ventricular contraction. According to the theory proposed in this paper, regurgitation might readily occur either before or following semilunar closure or during both periods. In cases of "premature systole," with the stethoscope applied over the heart, one frequently hears a short first sound, sometimes accompanied by an obscure murmur, followed quickly at times by a second sound of semilunar closure. The radial pulse wave belonging to the short ventricular systole is usually absent, but at the moment of systole there is a sudden bulging of the jugular bulbs, which forms a striking feature of the dyscrasia. An obvious explanation of the phenomenon is that the contractile wave which seizes the ventricular muscle fails to reach the distal extremities of the fibers; the resistance to outflow into the auricle is, therefore, less than that into the aorta and, accordingly, most of the ventricular content is regurgitated. The fact that the aortic resistance, and doubtless that in the pulmonary artery as well, rises steadily from minimal to maximal pressure between the beginning and the end of ventricular outflow, may possibly determine the failure of the weaker part of the cardiac muscle toward the end of systole. Hewlett[11] would probably class such regurgitations as examples of the "positive venous pulse" dependent upon simultaneous contraction of auricles and ventricles. Even if these two chambers do contract simultaneously, of which there has been no evidence in the examples studied by me, such

[11] The Interpretation of the Positive Venous Pulse. Jour. Med. Research, October, 1907.

synchronism of action should be attended by regurgitation from the ventricle, because its systole begins while the auriculoventricular valves are still open. But, as demonstrated by Mackenzie in his chapters on irregular heart action, the venous pulse in such cases can only be interpreted in the light of an exact conception of the relative time phases of auricle and ventricle, a subject which cannot be entered into here.

Before concluding this important subject it is well to picture again the condition of hydrostatic pressure existing within the ventricle toward the end of ventricular systole. The whole mass of the organ is in strong contraction. The auriculoventricular valves are held down at an indeterminate level by the pull of the papillary muscles. Suddenly, now, the outflow through the pulmonary artery ceases and, *provided there remains in the ventricle a sufficient residue of blood*, there is just as suddenly imparted to the under surface of the auriculoventricular valve an excess of pressure proportional to the relief of strain which has been cut off by closure of the semilunar valves. It seems wholly probable that the valve should thereby be pushed more or less into the cavity of the auricle and impart to its contained blood an impulse. As has already been shown, such an impulse could give rise to a wave in the venous tracing without necessarily involving any considerable displacement of blood mass. It is most significant that the more nearly normal the subject in whom the venous pulse is recorded, the more exactly does the beginning of the *v* wave correspond with the moment of closure of the semilunar valves. Now let us conceive the course of events under conditions of weakness of the papillary muscles. The resistance to ventricular outflow increases as the systole progresses and arterial pressure rises. This heightened hydrostatic pressure is imparted to the auriculoventricular valve, producing a strain whose total value is dependent upon the surface exposed. It should be expected that, were the tone of the papillary muscles to lessen toward the end of systole, still more, were the valve to become insufficient, we would find the *v* wave of the venous pulse to take its origin at a variable period preceding semilunar closure; and any sudden increase of the impulse imparted to the auricular contents, such as might be caused by a jet of blood escaping from the ventricle, could very well cause a doubling of the *v* wave such as is actually found in tracings of certain weak hearts.

From an *a priori* point of view it would seem probable that a safety-valve action of the auriculoventricular mechanism would be especially liable to occur at the beginning of ventricular systole when the ventricle is most distended and its contractile substance at the greatest mechanical disadvantage. It would seem that any undue retardation of the wave of contraction might permit such stretching of the papillary muscles as to allow regurgitation of blood during the "period of rising tension." The *c* wave, which covers most of the

period of outflow from the ventricle, should be modified by such regurgitation, but the factors entering into its production are still too uncertain to permit conclusions to be drawn from its change of form. It may be said, however, that in many of my tracings from cases of functional heart weakness, the magnification in the height of the *c* wave is very suggestive of valvular insufficiency early in systole.

The so-called safety-valve action of the auriculoventricular trap-door, whatever its significance in the cardiac economy, is generally regarded as an attribute of the right ventricle alone. If there be any truth in the proposition advanced above, that insufficiency of the auriculoventricular valve may occur through stretching of inert papillary muscles at the time of ventricular systole, there seems no reason why the same events should not, under appropriate circumstances, attend the action of the left ventricle. The anatomical fact that the tricuspid valve is suspended to a thick, circular ring of muscle, while the larger anterior flap of the mitral valve is made by a prolongation of the aortic wall, would lead to the expectation that functional dilatation of the auriculoventricular opening of the right side might easily occur, but not of the left. Tracings of the jugular pulse seem to offer experimental evidence of events occurring on the right side of the heart. One might expect that the record of movements of the left auricle, which can be obtained by connections with a rubber balloon resting in the œsophagus, should furnish certain evidence of mitral regurgitation. Yet Rautenberg,[12] in his observations on this subject, expressly states that in well-compensated organic insufficiency of the mitral valve the tracings of pressure changes and movements of the left auricle are almost identical with those derived from the normal subject. Yet speculation, if sharply defined as such, seems entirely excusable on so important a subject.

The distresss of an enfeebled heart is, in general, determined by the strain imposed by an excess of blood in one or more of its chambers. It is manifestly important for the pathologist to know the normal variations of the intracardiac blood pressure and the reactions of the cardiac chambers thereto. There can be little doubt that the movements of respiration normally produce a rhythmic variation of the ratio of intracardiac pressure on the two sides of the heart, yet definite physiological observations on this important subject seem to be wanting.

It is admitted that the inflow of blood to the right auricle is accelerated with the movement of inspiration and retarded with that of expiration. In inspiration the vascular bed of the lungs is increased and the resistance opposed to opening the pulmonic semilunar valves is probably diminished; but the aspirating force of inspiration

[12] Die Registrierung der Vorhofspulsationen von der Speiserohre aus. Deut. Arch. f. klin. Med., September, 1907.

would presumably be still more effective in retarding the blood flow into the left auricle; therefore, during inspiration we should expect an overfilled right heart and an underfilled left heart. The mechanical effect of an expiratory movement is just the reverse, and during expiration the diastolic content of the right heart would be decreased, while that on the left would be increased. An alternation of heart strain on the two sides would presumably run parallel with the degree of filling. A comparison of the curves of intrathoracic and arterial blood pressure gives general support to the foregoing position;[13] but the temptation to build upon even the most plausible theoretical notions is counteracted by the advance knowledge that the only trustworthy conclusion is to be reached by exact experimentation.

It is very desirable that we should have at hand means for the clinical estimation of the changing relations of heart strain. There is reason for supposing that such a means is to some extent provided in the *physiological* reduplication of the heart sounds.

These reduplications, whatever their cause, are synchronous with definite phases of respiratory movement. I have elsewhere pointed out[14] that a splitting of the second sound at the base of the heart, beginning early in inspiration and increasing in interval to the end of the movement, is a perfectly normal phenomenon. Reduplication of the first sound of the heart at the apex, although somewhat less frequent, is still extremely common. Potain long ago showed that this doubling is usually confined to the end of the period of expiration. I have already discussed this subject at length, concluding that reduplications of the first sound of the heart are a sign of somewhat excessive, but still physiological, heart strain.

Clinical observation shows that the reduplication of the first sound at the apex is usually heard only in the erect position. When the subject lies supine the reduplication disappears and is commonly replaced by a soft systolic murmur that might easily escape detection. I have ventured the opinion that the reduplication itself is due usually to a late contraction of the papillary muscles of the left ventricle causing a corresponding and isolated sound of valve-tension, and that *the systolic murmur in the recumbent position is due to a real valvular insufficiency from imperfect papillary contraction.* Although the evidences for this conclusion are far from satisfactory, they seem to harmonize from different points of view. If crucial proof of the correctness of the suggestion can ever be furnished, a common "safety-valve" action of the mitral mechanism will have been established. The *a priori* probability that the right ventricle, at least, is provided with the capacity for a safety outflow within the range of physiological action is greatly strengthened by

[13] Foster's Physiology, 4th ed., 364.

[14] The Clinical Significance of Reduplication of the Heart Sounds. AMER. JOUR. MED. SCI., 1898, cxvi.

the clinical study of persons who manifestly suffer from functional insufficiency of the heart and in whom tracings of the jugular pulse furnish the most constant objective sign of the cardiac weakness.

We turn now from a consideration of the problematical functional insufficiency of the auriculoventricular valves, the purpose of which in the economy must be to save the ventricles from overstrain, to a similar provision looking to the safety of the auricles. Admitting that the so-called "negative" venous pulse may at times have its origin in mere rhythmic intermission and acceleration of blood flow into the right auricle, there seems in many cases no doubt of the truth of Mackenzie's contention that even the "negative" or "auricular" form of venous pulse represents imperfect closure of the mouths of the great veins at the time of auricular systole, allowing regurgitation of blood from the contracting auricle. The important conclusion, deduced by Mackenzie from his clinical studies, that the existence of such a venous pulse always postulates a "limitation of the field of response" of the heart, almost necessarily carries with it the corrollary that even the lighter forms of venous pulse are manifestations of a *compensation* through which overstrain of the delicate auricle is prevented through backflow into the capacious "venous reservoir." A consideration of the conditions in compensated mitral disease leaves little doubt that the left auricle enjoys, at least, as efficient a protective outlet as the right.

In conclusion, while admitting the lack of the essential crucial evidence for the positions assumed in this paper, the physician in his practice must often act with less secure support. Strength has been added, I trust, to the conception that regurgitation from the auricles, thus preventing overstrain, is an occurrence so frequent that it cannot be regarded as a pathological event. Also, the indications are unmistakable that regurgitation from one or both ventricles, not caused by excessive dilatation of the auriculoventricular rings, but due to valvular insufficiency produced in another way, is an early and habitual sign of heart weakness. The therapeutic importance of the recognition of such signs of enfeeblement hardly needs an advocate.

CARDIAC ARRHYTHMIA.[1]

FROM A PRACTICAL STANDPOINT, IN THE LIGHT OF RECENT INVESTIGATIONS.

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Before entering upon a description of the various forms of cardiac arrhythmia it may be profitable to review hastily a few of the basic tenets of the myogenic theory of cardiac activity, since the modern studies of arrhythmia are based upon this conception. The myogenic doctrine maintains that the rhythmic contraction of the heart is due primarily to inherent properties of the cardiac muscle, and relegates to a position of secondary importance the nervous and chemical stimuli which doubtless play a part in the regulatory mechanism.

The heart muscle possesses five distinct properties, which may vary independently of each other: automatic stimulus production, conductivity, irritability, contractility, and tonicity. By *chronotropic influences* are meant those which influence the production of motor stimuli; when positive they accelerate stimulus production, shortening the time required, and thus increasing the rate; when negative the opposite effect is produced. When primary, stimulus production is accelerated directly, when secondary, the results are brought about through changes in irritability or conductivity. *Dromotropic influences* affect conductivity; *bathmotropic*, irritability; and *inotropic*, contractility. For example, stimulation of the pneumogastric nerve produces arrest of the heart. This may result from cessation of stimulus production (primary negative chronotropic influences), or through depression of excitability (primary bathmotropic influence), or to the fact that the stimulus though normally generated and received is not conducted (negative dromotropic influence).

The heart muscle has the ability to generate rhythmic stimuli, the nature of which is at present unknown, though a chemical character has been suggested, and not without strong reason, for recent experiments have demonstrated the necessity of certain inorganic salts in the maintaining of heart rhythm. Normally these stimuli arise in the venous ostia of the right heart, and pass downward through the bundle of His to the ventricles, not, however, as the result of nervous conduction, but by reason of muscular continuity, from cell to cell (Gaskell, Engelmann). Stimuli, however, may be generated at

[1] Read at a meeting of the College of Physicians of Philadelphia, April 1, 1908.

other points, but under normal conditions they are not of sufficient intensity to interfere with the rhythm, as may be the case pathologically, when the route of conduction may even be reversed. The left auricle, however, seems to have no power of automatic rhythmicity. The wave of contraction passes through different parts of the heart at a varying rate, a condition which favors a more complete ventricular systole (Engelmann). Stimulus production and conduction, as well as contractility, are much influenced by the phase of the cardiac cycle, the function of each being depressed during systole, and gradually restored during diastole. All these functions may vary in activity, independently of each other; they are also much influenced by the pabulum supplied by the arteries and lymphatics, by the removal of waste products through the veins, and by nerve impulses. Stimulation of the vagus depresses stimulus production, conductivity, and contractility. Stimulation of the accelerator nerve has the opposite effect. The vagus acts very slightly if at all on the ventricles, except indirectly through the auricles (Erlanger). In complete heart block the administration of atropine, though increasing the auricular rate, will have no effect on the ventricular rhythm (Gibson and Ritchie). Heart block itself may result from vagus action; this, as has been shown by Mackenzie may occur during deglutition. Conductivity is depressed by asphyxia; and when this function is below par the administration of digitalis still further increases this depression, a practical fact of great clinical importance to which I shall again allude.

From the foregoing brief review it is easy to see that in order to explain satisfactorily and study the different forms of arrhythmia we must first reduce them to their simplest terms, so to speak—that is classify and group them according to whichever one of the five fundamental properties of the heart muscle is chiefly or entirely concerned in their production.

Experimental physiology has made such rapid strides of late years that it is well nigh impossible for the general practitioner of medicine to keep pace with its latest developments, but our knowledge of the physiology of the circulation has undergone such extensive and important basic changes of late (through the labors of Gaskell, His, Erlanger, Engelmann, Hering, Mackenzie, Cushny, Wenkebach, and others), changes which are daily receiving corroboration and endorsement, that the physician can no longer afford to be uninformed of at least their fundamental principles. The myogenic theory of cardiac activity, while not as yet accepted by all physiologists, is daily gaining adherents. Although it doubtless still requires an enormous amount of investigation and revision, nevertheless, it elucidates satisfactorily and with such minute exactness so many hitherto unexplainable conditions, such as Adams-Stokes disease, extrasystolic arrhythmia, etc., that its acceptance as a working basis is practically necessitated. It was in a very similar

manner that Wasserman's theory regarding the blood complement, etc., led to a much more accurate and satisfactory knowledge of the question of immunity than had previously obtained.

The first type of pulse irregularity to which I wish to call attention is:

1. JUVENILE ARRHYTHMIA. It is, of course, well known that respiratory activity influences the pulse. In normal individuals inspiration increases and expiration decreases the rate. Some people show this phenomenon much more than others. It is especially common in youth, and has hence been called by Mackenzie the youthful type of arrhythmia. The type of irregularity affects chiefly the diastolic period, and for this reason it is noted especially when the pulse is slowing down, after infancy, and after a febrile attack. Mackenzie found that all children at some time exhibited this symptom. It occurs most frequently between the ages of eight and fifteen years, becoming less common toward puberty. It is pathological only when induced to a marked degree by quiet breathing, or when it is not controlled by apnœa. It indicates nerve irritability and is proof of vagus activity. It occurs in fevers, especially during convalescence, in neurasthenia, and in cerebral lesions causing irritation of the vagus. Tracings taken from the precordial region show that it consists of a true intermission, and is not simply a missed beat at the wrist resulting from extrasystole. Needless to say, it has an altogether entirely different genesis than the pulsus paradoxicus, which is entirely due to the mechanical relations of the pericardium and mediastinum. Peters has shown that the vast majority of arrhythmias occurring during the late stages of diphtheria and scarlet fever are of the respiratory type. These two infections are so frequently the cause of serious myocardial lesions that pulse irregularities following them are naturally regarded with alarm. If, however, in such a case we are able to demonstrate a simple respiratory arrhythmia, our prognosis will of necessity be much affected by the finding.

The second group of arrhythmias which I shall consider is the one most frequently encountered, namely:

2. EXTRASYSTOLE. As a rule, an intermittent pulse is due to extrasystoles, resulting from abnormal irritation at some point of the musculature, generally the ventricle, although it may result from heart block or diminished excitability. These extrasystoles can often be heard on auscultation and are registered on the cardiogram, although the pulse wave may not reach the periphery. The later the extrasystole occurs in diastole the louder will be the sound heard, and the larger the pulse wave, so that if it occurs shortly before the normal time we may get the simulation of a normal, somewhat anticipated, systole. The futile contractions (frustrane Kontraktionen) of Hochhaus and Quinke are examples of extrasystoles occurring early in the diastolic period. Extrasystoles may

also occur without causing dropped beats or compensatory causes, in cases of bradycardia in which the refractory phase is short in comparison with the length of the pulse rhythm. Experimentally an extrasystole may be produced by increasing the *vis a fronte* in the circulation; it is also seen in digitalis poisoning, and after the administration of calcium. It may also be produced by mechanical and electrical means.

Extrasystoles are explained by the fact that as the refractory phase of the heart rhythm is coming to a close the muscle becomes more and more susceptible to stimuli. An abnormal stimulus sets off the contraction before its time, just as hot carbon in the cylinder of a gasoline engine explodes the charge before sufficient compression has occurred. As the result we see a weak, early contraction which, owing to an insufficient volume of blood and inadequate contractile force—resulting from shortened disatole—barely, if at all, opens the aortic valves. This extrasystole, however, throws the heart into a second refractory period at a time when the normal physiological stimulus should occur; in this manner a beat is dropped and a compensatory pause is brought about. By reason of the latter the number of heart beats per minute may remain unchanged despite the occurrence of extrasystoles, and the length of time occupied by the normal contraction and the extrasystole plus their respective pauses may occupy an equal interval of time as would two normal contractions. This condition of affairs is characteristic of extrasystoles arising in the ventricle. When the abnormal stimulus arises at the mouths of the veins in the right heart, the compensatory pause is absent; if in the auricle itself, it may or may not be present; if in the ventricle, it is present and is not infrequently prolonged.[2] But if this prolongation is great we are strongly led to suspect that the trouble is due to depression of conductivity and not to the fact that the stimulus arrived in the refractory period.

A series of extrasystoles following each other gives rise to the pulsus bigeminus, trigeminus, quadrigeminus, etc., according to their frequency, while a continuous series may produce a clinical paroxysmal tachycardia, although Hoffmann has shown that in tachycardia due to this cause a series of compensatory pauses may be distinguished.

Extrasystole is often recognized subjectively by the patient as a dropped or a doubled beat, or a powerful thump which follows the compensatory pause. It is not however, always an easy matter to determine whether an arrhythmia is or is not due to extrasystole. Thus, when the intermissions occur in different kinds of groups, with varying degrees of peripheral pulsation; when we find abnormally long compensatory pauses, and small pulses following them, we may be unable to make out the regularity of the rhythm both

[2] Hering, Pflüger's Arch., lxxxii; Präger med. Woch., xxvi, Nrs. 1 und 2.

in the ventricle and in the artery, despite the fact that the stimulus production may be going on normally at the venous ostiæ. Furthermore, extrasystoles often complicate other forms of arrhythmia, making interpretation difficult. We have the three definite varieties of extrasystoles: (1) Auricular; (2) ventricular; and (3) auriculoventricular; the latter two being the more common. What is known as the interpolated ventricular extrasystole is one which has but little influence on the rhythm, although it may produce a retrograde auricular systole.[3]

Extrasystole is seen in many conditions, which may be grouped as follows: (1) Physiological or individual; (2) after severe exertion; (3) in women at puberty, in pregnancy, and at the climacteric; (4) toxic causes: alcohol, ether, tobacco, infectious fevers, especially during convalescence, and in intestinal disorders; (5) nervous disturbances: neurasthenia, hysteria, neuroses; (6) arteriosclerosis; and (7) organic heart disease.

Clinically, extrasystoles are generally manifested as two or more approximated beats, followed by an intermission. If the extrasystole occurs early in the diastolic period, as is generally the case, we, as a rule, hear on ausculatation a muffled third sound during diastole. The heart is at this time insufficiently filled with blood, the pressure in the aorta is still high, and the ventricular contractile power weak, owing to insufficient rest, as a result of which the aortic valves are not opened by the extrasystole. If the extrasystole occurs later in the diastolic period, a second sound may be produced. The extrasystole is generally followed by an unduly prolonged silence, which is, in turn, brought to a close by a strong, loud, muscular sound of the next regular systole. If the extrasystole has resulted in opening the aortic valves, a small, early pulse wave is seen on the sphygmogram—pulsus bigeminus. Rarely the tracing may simulate the normal dicrotic wave.

Extrasystoles may be induced by a number of causes. It has been shown, both experimentally and pathologically, that increased aortic blood pressure is a prolific source of this symptom. Extrasystoles are also brought about through pathological changes in the myocardium, which appear to initiate a stimulus to which the neighboring normal muscular fibers respond. Increased excitability—positive bathmotropic influences—toxic or thermic in origin, are also often productive factors. And finally, we have to include nervous influence. The latter influence is an indirect one acting through vasoconstriction, which is a well-known excitant. Extrasystoles often occur as an early sign of functional derangement in patients with obesity or arteriosclerosis, and although they may exist for years without any more serious symptoms, their presence calls for careful investigation of the case in which they appear. According to

[3] Hering, XXIII Kongr. f. Inn. med., München, 1906.

Hewlett, extrasystoles usually do not develop during the acute myocardial lesions of infectious diseases, but when they do appear under such conditions they are of a relatively worse significance.

The occasional occurrence of extrasystole in young, otherwise healthy individuals is of little importance. The symptom, however, always points to cardiac irritability, and while this may be normal for some individuals, its presence should always make us look carefully for intracardial and extracardial abnormalities. Anyone who has auscultated the heart in horses or dogs is aware how frequently extrasystoles occur, and how easily they are produced in these animals. Similarly there are certain individuals, notably those with highly strung nervous systems, who exhibit the symptom more or less constantly. Beyond indicating cardiac irritability, extrasystoles give us no positive clew; they are not pathognomonic of any particular disease.

Wenkebach divides individuals exhibiting extrasystoles into four groups:

1. People who live to old age without demonstrable heart lesion, who have experienced this symptom off and on for a variable period of years, without any deleterious consequences. Such cases probably result from a congenital or acquired hypersusceptibility to stimuli.

2. The extracardial group, to which class belong the toxic and reflex cases. Extrasystoles are very commonly seen during the convalescence from infectious fevers—typhoid fever, pneumonia, diphtheria, etc. They also occur as a result of the use of tobacco, alcohol, tea, and coffee; as well as in cases of gastro-intestinal disturbances, intestinal parasites, etc.

3. To this class belong those who are the subjects of circulatory disease, regardless as to whether the heart be seriously affected or not. For instance, cases with arterial hypertension frequently exhibit extrasystoles. The same may be said of arteriosclerotics. In these cases the symptom is often intermittent and readily produced by physical effort or mental excitement, Some of these cases can, of course, be explained on the basis of hypertension, but Wenkebach favors the view that the cardiac muscle becomes more irritable as age advances, for the following reasons: (*a*) Other signs of irritability, such as delirium cordis, are frequent in elderly people. (*b*) In both young and old subjects extrasystoles are of common occurrence when arterial tension is low. (*c*) Diseases in which hypertension is most frequent, such as chronic nephritis, are not those in which extrasystoles are most common.

4. Cardiopaths. In this class extrasystoles are of frequent occurrence. We can, however, draw no definite deductions by the mere fact of their presence as to the stage, character, or extent of the heart lesion, Productive factors are present in great numbers in such cases, that is, dilatation, inflammation, local cardiac anemia, overexertion, etc. The worst cases of heart disease often terminate

fatally without having at any time exhibited extrasystoles. Wenkebach concludes: After all that has been said, clinicians ought not to attach too much significance to extrasystoles in themselves, and yet they ought to consider it worth while to examine every case and determine whether a cardiac lesion is present or not, whether there are any conditions (and they must often be looked for outside of the heart) present, which could account, directly or indirectly, for the presence of the extrasystoles.

Mackenzie, basing his views on the postmortem histological findings of Keith, states that in the majority of cases of extrasystole in the aged there are to be found minor pathological changes in the auriculoventricular bundle which can account for the depressed conductivity and hyperexcitability, although he admits that there were also present abnormalities of the coronary arteries and of the ventricular musculature.

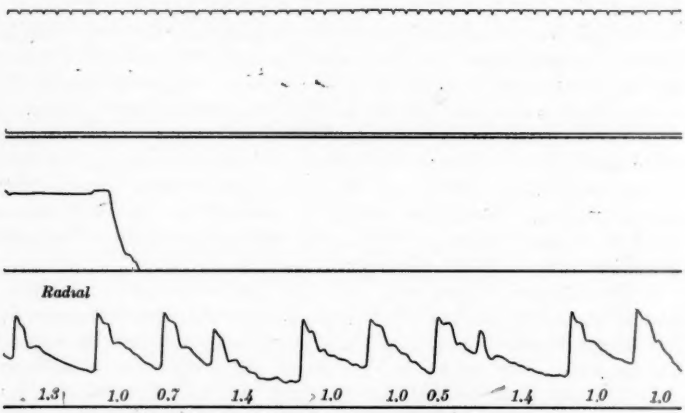

FIG. 1.—Extrasystoles of ventricular origin, followed by compensatory pauses. The normal systole plus the extrasystole occupy the same or a slightly longer interval of time than two normal beats.

The accompanying illustration (Fig. 1) shows extrasystoles of ventricular origin occurring in a patient with arteriosclerosis. It was present only temporarily after a change of posture. It appeared in the erect and in the recumbent position for a few minutes after the posture was assumed, and then gradually ceased. The systolic blood pressure was 135 mm., the diastolic 95 mm.[4]

Fig. 2 exemplifies the occurrence of extrasystoles of auricular origin. They are preceded by auricular (*a*) waves in the jugular tracing, and are not followed by compensatory pauses, as are ventricular

[4] The time recorded on all of the following tracings is in units of one-fifth of a second.

extrasystoles. This tracing was taken from a man aged sixty-six years, with a high grade of arterial thickening, and a systolic mitral murmur which completely replaced the first sound of the heart. The heart dulness was greatly enlarged to both right and left. The systolic pressure was 160 mm., the diastolic 110 mm. He complained of vertigo after any sudden change of posture, precordial oppression with dyspnœa on exertion, and improved under the administration of digitalis. There is marked delay in conductivity —the *a* to *c* interval being 0.4 instead of 0.2 of a second, indicating myocardial involvement.

Fig. 3 illustrates extrasystoles of auriculoventricular origin; in other words, the abnormal stimulus arises in the bundle of His. The large waves marked *a* and *c* are due to the simultaneous con-

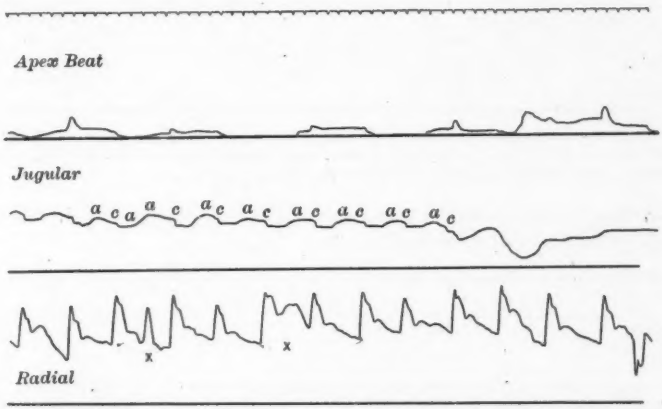

FIG. 2.—Extrasystoles of auricular origin. They are preceded by auricular waves in the jugular tracing *a*, and are not followed by compensatory pauses.

traction of the auricles and ventricles, both being premature. These waves are not followed by a *c* wave, and the diastole following is of the same length as the normal auricular rest period. This is the form of extrasystole which has been designated by some observers as the retrograde extrasystole, it being assumed that the abnormal stimulus arises in the ventricle and is transmitted backward to the auricle. This explanation is hardly satisfactory, however, for, as Mackenzie has pointed out, if such were the case there should be a delay of one tenth of a second in the appearance of the auricular wave—the time required for the stimulus to travel across the auriculoventricular bundle—whereas, in reality, the contraction of the auricle and ventricle are exactly synchronous.

This tracing was taken from a physician aged sixty-six years, who complained of precordial oppression, and a thumping in his chest

which occurred with each intermittence of the pulse. Physical examination disclosed a double aortic lesion. The radial pulse on

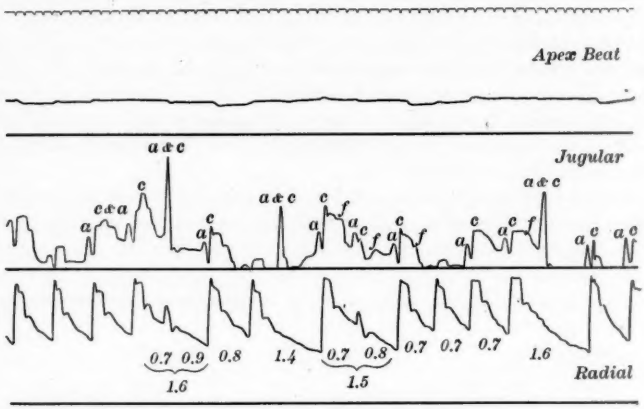

FIG. 3.—Extrasystoles of auriculoventricular origin (arising in the bundle of His). The tall waves *a* & *c* are due to the simultaneous contraction of the auricles and ventricles. They are not followed by *c* waves or by compensatory pauses.

the tracing shows the characteristically rapid collapse which follows the straight, high, systolic limb.

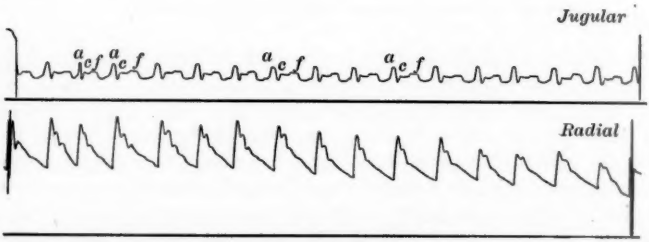

FIG. 4.—The same case as Fig. 2. The extrasystoles have disappeared. The jugular tracing shows the three normal waves.

Fig. 4 was taken from the same patient one year later. The arrhythmia had entirely disappeared. The pulse rate was 68, the systolic pressure 190 mm., the diastolic 110 mm., and the patient was comfortable and attending to his work.

Fig. 5 illustrates extrasystoles of ventricular origin, appearing early in diastole, followed by compensatory pauses, and failing to appear in the radial pulse, except occasionally, this being due to the fact that the ventricle has been insufficiently rested and inadequately filled with blood, as the result of which the aortic valves were barely if at all opened. This tracing was taken from a patient with failing compensation—a double mitral lesion, with marked cardiac dilatation; death occurred about two weeks later. On auscultation two fairly loud, close-coupled sounds were heard over the apical region, followed by only one second sound at the base (occasionally two sounds were heard at the aortic area, the second being muffled and indistinct). The pulse at the wrist was for the most part quite regular, but only one beat was felt, while two sounds were heard at the apex area. If the ventricle had been stronger we should have had a typical pulsus begeminus in the radial.

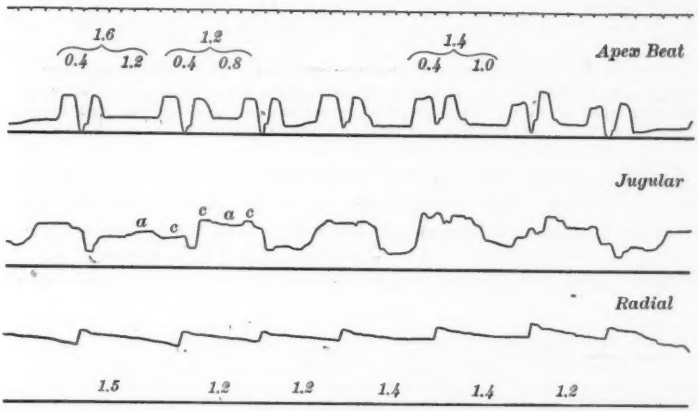

FIG. 5.—Extrasystoles of ventricular origin, which are manifest at the apex beat but do not reach the periphery. Only every other beat causes a radial pulse, thus producing a false bradycardia which without the tracing might have been construed as due to heart-block.

Fig. 6 shows a series of extrasystoles occurring in groups of two, producing a pulsus trigeminus, which occurred in a case of acute endocarditis, which terminated fatally about ten days later. At autopsy in addition to cardiac hypertrophy and ditatation ulceration of the aortic valves was found.

The following tracings illustrate most graphically the way in which digitalis improves the character of the pulse by suppressing the extrasystoles, producing a longer diastole, a more complete period of rest, and resulting in a greatly improved state of the circulation. Fig. 7 was taken from a case of mitral stenosis and insufficiency, with cardiac dilatation and tricuspid insufficiency. There

was general anasarca, cyanosis, orthopnœa with blood-tinged expectoration, and a pulsating liver. The tracing shows the marked

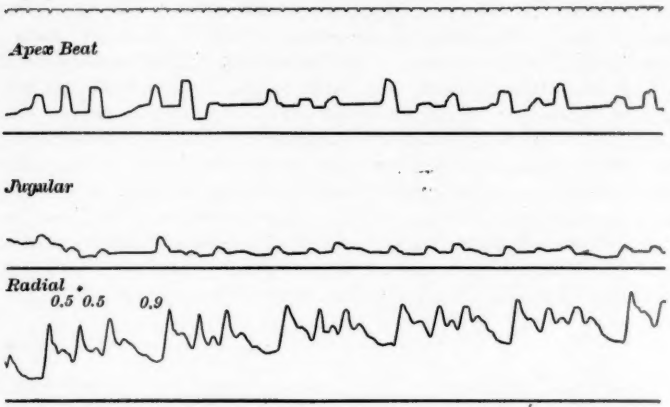

FIG. 6.—Pulsus trigeminus, resulting from extrasystoles occurring in groups of two. The jugular pulse is of the positive type—the waves are synchronous with the ventricular systole; taken from a case of mitral and tricuspid insufficiency.

arrhythmia complicated by extrasystoles, and a positive venous pulse. The patient was bled ten ounces and given three drams of the infusion of digitalis thrice daily. A few days later the general condi-

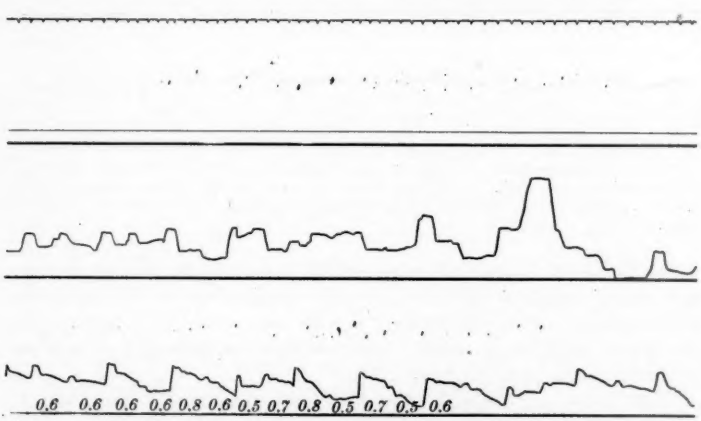

FIG. 7.—Double mitral disease with tricuspid insufficiency, showing extrasystoles and a positive venous pulse.

tion was greatly improved. At this time the second tracing was taken (Fig. 8), which shows a much slower, better filled and sustained

radial pulse, and many fewer extrasystoles. It will be seen that the jugular pulse is still of the positive type resulting from the tricuspid leakage.

The next type of pulse irregularity to which I would call attention is a variety which is met with quite frequently in the advanced stages of heart disease, generally with failing compensation:

3. PERPETUAL ARRHYTHMIA. Perpetual arrhythmia is one in which no regular rhythm whatever can be made out for long periods of time—weeks or months. The subject of its etiology requires further elucidation. When present in its typical form three characteristics are noted: (1) The jugular tracing shows no auricular wave; (2) it is always associated with a positive venous pulse; (3) absolutely no underlying rhythm can be detected. As to the second

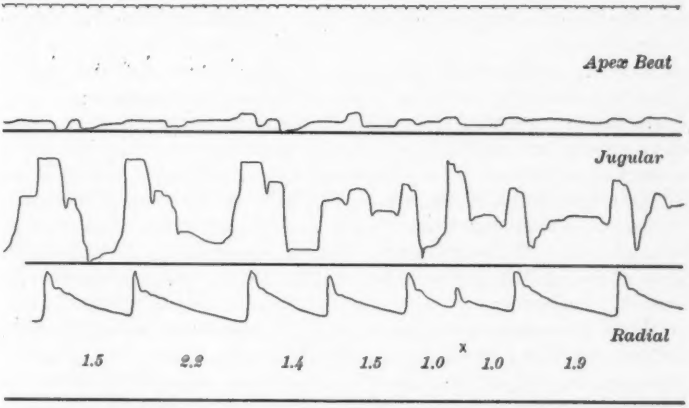

FIG. 8.—Tracing from the same patient as Fig. 7, after venesection and digitalis. Most of the extrasystoles have dropped out. There is a marked improvement in the character of the radial pulse, and there was a corresponding amelioration in the patient's condition. x, extrasystole.

qualification—the positive venous pulse—the majority of authorities hold that this is due to tricuspid insufficiency or to paralysis of the auricle. As to the etiology of this variety of irregularity, Wenkbach believes that it is due to a block between the sinus and the auricle, the result either of muscular weakness or depression of conductivity, and not to auriculoventricular extrasystole or to pathological changes in the auricular muscle. Erlanger has recently performed some experiments which prove the possibility of an experimental sino-auricular block.

When a tracing from a case of perpetual arrhythmia is examined we are struck by the fact that no two pulse waves are of equal length. Clinically the condition is often spoken of as delirium cordis, but as Hewlett has recently pointed out, this character of pulse is sometimes met with even if the pulse is slow. We have

already mentioned the fact that a permanently irregular pulse without tachycardia or bradycardia, unaffected by respiration, digitalis, or atropine, is always associated with a ventricular venous pulse. Since auricular paralysis or tricuspid insufficiency is practically only encountered in cases with broken compensation, we find the permanently irregular pulse only in very serious cases. It is apparently always intracardial in origin, and it is often met with in arteriosclerosis and myocardial disease. Minor degrees of this irregularity may resemble the youthful type; the severer forms are often complicated by extrasystoles. The beneficial effects of digitalis in these latter forms are owing to the fact that this drug, by depressing the function of conductivity in the auriculoventricular bundle, causes a dropping out of the extrasystoles. Perpetual arrhythmia may appear and disappear either suddenly or gradually; in the latter case we often see a reappearance and steady increase in the size of the auricular wave.

Another variety of arrhythmia which has lately come into prominence as a pathological entity of much clinical importance is:

4. Heart Block. This class of arrhythmias consists of those cases which are due to depression of conductivity (heart block), a condition which may exist in varying degree. It may be manifested by a dropping out of the ventricular beat or by a complete dissociation of auriculoventricular rhythm. The most marked and typical cases are those of the Adams-Stokes syndrome, in which the auricles beat more rapidly than the ventricles. It has been shown that at least the majority of these cases are due to a lesion in the bundle of His. If a sufficient number of muscular fibers in this bundle are destroyed to prevent completely the conduction of the stimulus from the auricle to the ventricle, the latter chamber initiates its own rhythm, beats independently, and a complete heart block is said to exist. If, on the other hand, every second, third, or fourth auricular contraction is able to set the ventricle in action, the condition is termed an incomplete block. Depression of conductivity sometimes occurs temporarily in the convalescent stage of diphtheria, typhoid and rheumatic fever. According to Huebner, the bradycardia, occurring after typhoid and other infections is the result of a toxic depression of conductivity. Heart block may also be produced by digitalis, and when already present the administration of this drug still further depresses the function of conductivity, and is therefore absolutely contra-indicated. This effect of digitalis is not necessarily the result of vagus stimulation, for the auricular rate may be unaffected, although the ventricular beats fail; and the pneumogastric nerve seems to act on the latter chamber only through the former. It has been shown that the pneumogastric nerve can produce not only chronotropic, but also dromotropic and inotropic effects (Muskens, Hoffmann, Engelmann). Digitalis seems to exert a specific local action on the function of conductivity.

The existence of delayed conductivity can only be determined by the coincident tracing of an arterial and a venous pulse (usually the radial and the jugular are employed). In normal cases the *a* to *c* interval as it is called—that is, the time which elapses between the systole of the right auricle and that of the left ventricle—should be not more than 0.2 seconds with a pulse rate of 70.

If this time is much increased, conductivity is impaired. If the block is complete, the auricular waves in the jugular and the carotid waves bear no constant relations to each other. Experimentation and clinical observation have shown that depression of conductivity may occur as a result of local or general anemia, of toxemia, of degeneration of the cardiac muscle, especially fibroid degeneration and myocarditis of the auriculoventricular groove. Fatigue may be a precipitating factor. I have taken tracings of a case which showed a partial block after exertion which disappeared after a few minutes rest in the recumbent posture.

Lommel has already called attention to intermittent vagus activity resulting from fatigue.

From a clinical standpoint it is important to decide whether a heart block is nervous or muscular in origin, since the former is much more amenable to treatment, and of less serious prognostic import. In attempting to determine this question we must not forget that heart block may be brought about by secondary dromotropic effect of other disturbances, such as a high pulse rate, which does not allow sufficient time for a restoration of functional activity.

In the heart block due to muscular causes the pulse rate is generally lower than in the nervous form. In the former instance the pulse is regularly intermittent, in the latter variably so, and much influenced by respiration. If the administration of a physiological dose of atropine has no effect upon the heart block, the vagus factor has been eliminated.

Heart block may occur between any two parts of the cardiac musculature possessing spontaneous rhythmicity if the bridge of connection be severed or the passage of stimulus be interfered with.

Some tracings published by Gibson seem to indicate that heart block may also be caused by depression of excitability.

Fig. 9 was taken from a case of Adams-Stokes disease. It represents an incomplete heart block with a three-to-one rhythm. The ventricle responds only to every third auricular stimulus. The function of auriculoventricular conductivity is much depressed, the *a* to *c* interval lasting four-tenths instead of two-tenths of a second. Death occurred about three months after the first attack of vertigo.[5]

Fig. 10 is a tracing from another case of Adams-Stokes disease, occurring in a man of middle age, who has been under observation

[5] Case referred by Dr. J. W. Irwin. A full account, with histological examination, will soon be published.

for three years, at which time he was admitted to the Philadelphia General Hospital complaining of dyspnœa on exertion. Tracings

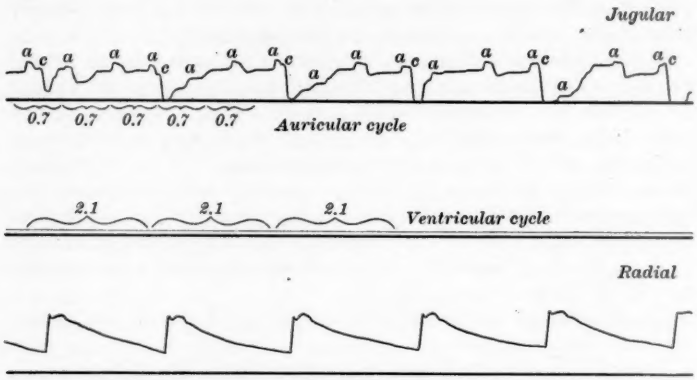

FIG. 9.—Adams-Stokes disease, with a three-to-one rhythm. There are three contractions of the auricle to one of the ventricle.

made three years ago show the same kind and degree of heart block, but since that time he has been unable to work, and vertigo as well

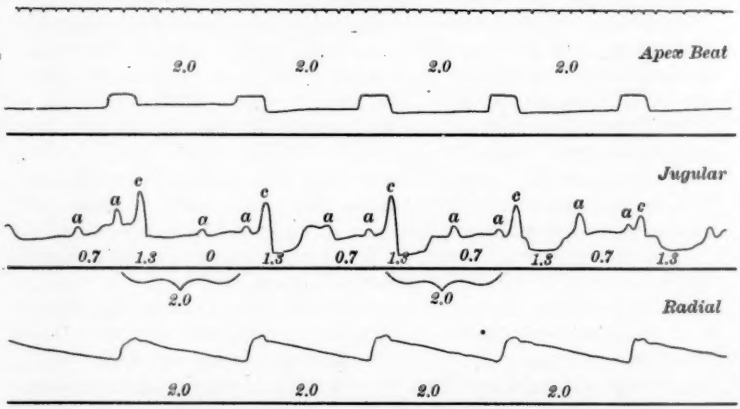

FIG. 10.—Adams-Stokes disease, with a two-to-one rhythm.

as syncope have been added to his list of symptoms.[8] At the time this tracing was taken his systolic pressure was 145 mm., the dias-

[8] I am indebted to Dr. F. B. Stahl for the opportunity of making the second series of tracings from this case.

tolic 80 mm. The radial pulse was 30 per minute, both erect and recumbent. It was unaffected by respiration, exercise, or the administration of atropine.

Fig. 11 is from another case of heart block with a two-to-one rhythm occurring in a man, aged fifty-eight years, who complained of dyspnœa on exertion, precordial pain radiating to the arms and head (occiput), with vertigo of slight degree. He was the subject of advanced arterial degeneration and had marked hypertension. The highest figures recorded were 235 mm. for the systolic, and 140 mm. for the diastolic pressure. At the time this tracing was taken the pressures were 150 mm. and 100 mm., respectively while recumbent, and 175 mm. and 110 mm. while erect. The radial pulse erect was 70; recumbent, 54 per minute.

It would seem, therefore, that we were dealing with a case of heart block in the early stages, since the pulse rate was still affected by change of posture. On auscultating over the body of the heart,

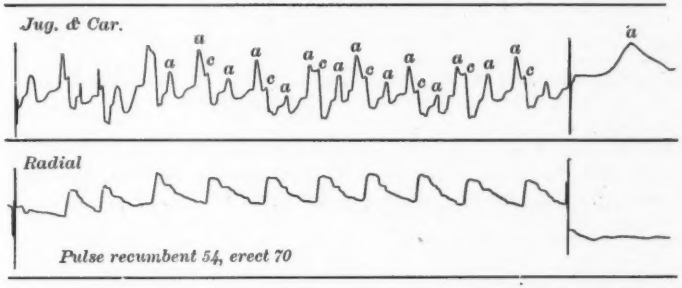

FIG. 11.—Auriculoventricular heart-block, occurring in a case of arteriosclerosis with marked hypertension and symptoms of angina pectoris. Two-to-one rhythm.

two clear, muscular sounds were heard in addition to the aortic second sound, which was high pitched, clear, and ringing, during which time only one pulse beat appeared at the wrist. Furthermore, every other auricular contraction was followed by a ventricular response. The radial pulse shows the characteristics of arterial hypertension.

Fig. 12[7] is a tracing taken from a man of about fifty years of age, a business man of the modern "hustler" type, in June, 1907. He complained of some dyspnœa on exertion, and an examination of the chest revealed the physical signs of aortic insufficiency, mitral insufficiency, and a dilated hypertrophy. He had lost twenty-five pounds in weight within one year, and had taken three series of Nauheim baths in this country. The radial tracing shows the typical large-waved, collapsing pulse of aortic insufficiency, with

[7] Case referred to me by Dr. J. H. Musser, as was also the following case.

rather well-marked dicrotic wave, perhaps the result of the mitral insufficiency. He had no fever. The jugular wave is of relatively normal proportions, and shows no evidence of auriculoventricular

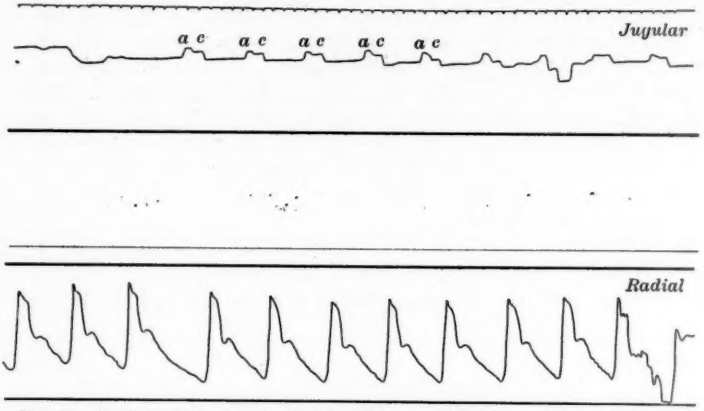

FIG. 12.—Aortic insufficiency and mitral insufficiency. Normal coördination of the auricles and ventricles is shown by the jugular pulse.

dissociation. Every auricular wave is followed by a ventricular wave. The blood pressure was 140 mm. systolic, 90 mm. diastolic. During the summer his pulse rate fell to as low as 37 per minute,

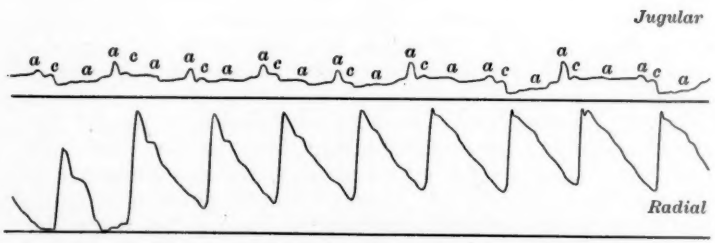

FIG. 13.—The same case as Fig. 12, taken five months later. Distinct heart-block with a two-to-one rhythm.

and for a period of six weeks remained between 37 and 40, gradually getting up to 60. During this time he felt restless and weak, but had no vertigo or syncope. On resuming his active career he found that

when physically tired his pulse would beat at 40, but that rest in the recumbent posture would, in a short time, restore the rate of 60. In November, 1907, I had another opportunity of making a pulse tracing (Fig. 13).

Here we are dealing with a distinct case of incomplete heart block, in which the rhythm varies between two-to-one, and three-to-one. The pulse rate was 48 per minute, and shortly after the preceding tracing was taken, the patient having been in the recumbent posture, returned to 60 with a disappearance of the heart block, as is shown in Fig. 14.

The last two cases described are, I believe, the first instances recorded of what with strong probability appears to be the early developmental stages of Adams-Stokes disease. How long patients afflicted with this malady may live probably varies greatly under

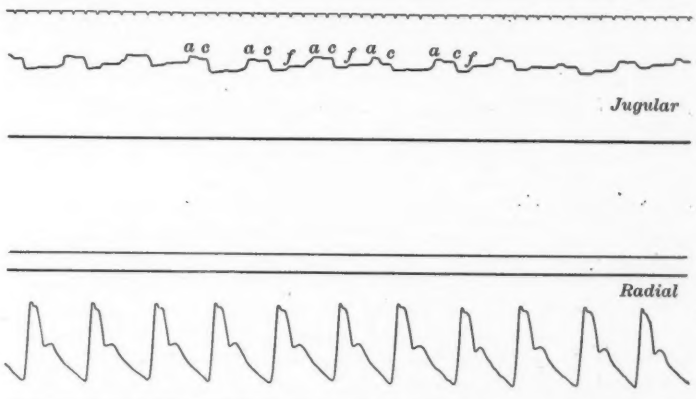

FIG. 14.—The same case as the two preceding figures, after rest. The heart-block has disappeared, and the normal auriculoventricular rhythm has been restored.

different circumstances, being dependent of course on the extent of damage done to the auriculoventricular bundle, and the character of the lesion affecting it. The case from which tracing No. 5 was taken was known to have had heart block three years ago, and doubtless had had the condition for some time before he came under observation, since his lesion has been very slow in progressing. It is more than probable that careful study by cardiosphygmographic means of many cardiopaths will teach us that heart block is a much commoner lesion than we have heretofore been led to suppose. In fact, when we consider the anatomical position of the bundle of His, it is rather surprising that it is not more frequently damaged by inflammatory processes in its neighborhood.

Fig. 15 illustrates another case of heart block due to the administration of digitalis. In this instance the pulse rate dropped sud-

denly from 80 per minute to about 40. At the time this tracing was taken it was 38.[8]

The last form of arrhythmia remaining to be considered is the rarest variety, namely:

5. Depression of Contractility—Pulsus Alternans. In this class of arrhythmias are placed the cases which are inotropic in origin; that is, those due to depression of contractility. Contractility is easily measured, since cardiac contractions are always maximal if the stimulus provokes any contraction. They are characterized by the alternate appearance on the arterial syphgmogram of a large and a small wave, either with or without increased pulse rate. True cases of this condition are not the result of extrasystole, since they are not followed by compensatory pauses, nor are they due to depression of conductivity, as can be determined from the jugular tracing;

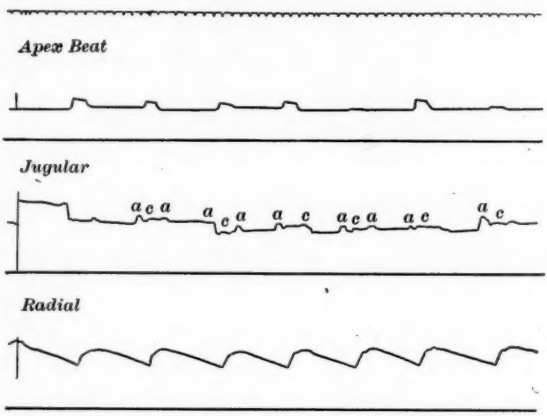

Fig. 15.—Heart-block precipitated by the administration of digitalis.

they arise from heart weakness. The heart while at rest may contract with regular force, but when increased effort is called for uniform contractions cannot be produced. The contraction is slowly accomplished and the muscle insufficiently rested by the time the next stimulus arrives. Hering has described as pulsus pseudo-alternans those cases which upon analysis prove to be the results of extrasystoles. Cases in which the smaller wave occurs too soon are extrasystolic in origin; if the diastolic period is normal, it may or may not have this origin, but if the small wave is retarded we have to deal with a true pulsus alternans. This form of arrhythmia is explained by Wenkebach as follows: With diminished contractile power, if a single normal stimulus happens to occur a trifle too early, it finds the myocardium in a still more hypodynamic condition than

[8] I am indebted to Dr. A. O. J. Kelly for the opportunity of studying this case.

did the previous stimulus. As a result, the succeeding contraction will not only be smaller, but will, as has been shown by Hoffmann, be performed more rapidly. If, then, the next stimulus does not occur too late, the longer pause will permit a more perfect restoration of function, and the next wave will again be large. This, in turn, being of longer duration and its pause therefore shortened, will be once more succeeded by a smaller wave.

Clinically, pulsus alternans is recognized by this peculiar, regular alteration of large and small beats. Characteristic of it is the fact that once established it persists for long periods of time, being thus in marked contradistinction to the cases of extrasystole which are irregularly intermittent, as a rule, and have never been known to occur steadily and regularly for weeks. Furthermore, extrasystoles when present tend to vary their appearance during different phases of the diastolic period. The difference in the size of the two waves of a pulsus alternans may vary within wide limits; generally we may assume that the amount of cardiac weakness is directly proportionate to the smallness of the second wave. It also often bears a direct relation to the rapidity of the rate—the more rapid the action the greater the alteration. Pulsus alternans is met with in severe myocardial and valvular lesions. It is a distinctly rare condition. Wenkebach states that he has encountered but two cases. The condition is met with in the arterial hypertension of nephritis and angina pectoris, but it sometimes occurs after infections (influenza), and in such cases may disappear.

Thus far we are in possession of but little knowledge concerning stimulus production and tonicity. Gaskell has shown that the latter property may be altered by various drugs, and it has been suggested that cardiac dilatation may result from depression of this function.

Prognosis. It is sometimes difficult to draw definite conclusions as to the prognostic importance of an arrhythmia. Of course, the age of the individual, his occupation, habits, and social condition, have to be taken into consideration. We can also obtain much valuable information from the patient's general condition, the state of the bloodvessels, arterial tension, pulse rate, etc., and especially from the effect of exertion upon these factors. We can rarely afford to prophesy on the basis of the sphygmograph alone any more than we can on the clinical thermometer and yet both of these instruments are useful adjuvants in diagnosis.

Arrhythmia of muscular origin is more serious than that due to nervous causes. As has already been pointed out, we can determine the presence of the latter by deep breathing and by the effect of the atropine test. Further, the time and cause of an arrhythmia will much modify our opinion of its seriousness. Thus, for example, extrasystoles occurring during the convalescence from infections have generally very little importance, whereas the appearance of this symptom during the fastigium is often of sinister import.

The respiratory type of irregularity is generally of trivial consequence. Extrasystoles, although often insignificant, require careful investigation, especially if occurring in middle-aged or elderly people. Perpetual arrhythmia is always serious, and the same may be said of that rare condition, pulsus alternans. Auriculoventricular heart block also is unfavorable, for although in some subjects the attacks may be precipitated by the injudicious administration of digitalis and disappear after withdrawal of the drug, and other subjects may live for a number of years despite its presence, the symptom always indicates a disturbance of muscular functionation.

THE PECULIARITIES OF THE SYMPTOMATOLOGY OF RHEUMATISM IN CHILDREN.[1]

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The etiology of rheumatism is still a subject for discussion, and, unlike such diseases as diphtheria and typhoid fever, its existence in any case is not susceptible of scientific proof. The claims of rheumatism to our consideration as a separate disease entity must be based, as in such diseases as bronchopneumonia, upon the specific character of the anatomical lesions, or, as in such diseases as scarlet fever, upon the specific character of the clinical picture. The peculiarities of the symptomatology of rheumatism are therefore important, not alone from their own intrinsic interest and their bearing upon the practical aspect of the disease in diagnosis and treatment, but also from the possible light which they may throw upon the unsettled question of an ultimate specific etiology.

One fact, indeed, is now generally recognized: that rheumatism is a disease that differs widely in its clinical manifestations and phenomena as we pass from earlier to later childhood, from childhood to adolescence, and again into adult life. I know of no disease common to children and adults which presents such a surprisingly different clinical picture at these two ages. I find that in beginning to demonstrate cases of rheumatism in childhood to students whose previous training has been confined to adults, its peculiarities impress them like the revelation of an entirely new disease.

In the adult, rheumatism presents itself as an acute disease characterized by inflammation in the joints, with its severe symptoms referable to the joints and with a tendency to cardiac complications

[1] Read by invitation at a meeting of the Philadelphia Pediatric Society, February 11, 1908.

of comparatively slight clinical significance in their immediate result. In the child, rheumatism presents itself as an acute disease characterized by inflammation in the heart or joints, or both, with its severe symptoms referable to the heart, and with a tendency to arthritic symptoms of comparatively slight clinical significance. This is the principal contrast, stated in its broadest form. In a more detailed view of rheumatism in childhood many further points of contrast, of great interest, are encountered.

As to the mode of onset of rheumatic fever, we find that in the adult the disease begins almost invariably with fever and arthritis, usually polyarthritis, characterized by pain, tenderness, redness, and swelling at the affected joints. In an analysis of 223 cases of rheumatic fever in children, I found that the mode of onset was as follows: Fever and arthritic symptoms, 88 cases; fever and cardiac symptoms, 82 cases; fever and both arthritic and cardiac symptoms, 25 cases; fever only, 18 cases; fever and sore throat, 6 cases; fever and chorea, 1 case; and unknown, 3 cases.

From this it appears that, although a little the most common mode of onset is with symptoms referable to the joints alone, an onset with symptoms referable to the heart alone is almost as common. By cardiac symptoms are meant not the evidence of insidiously developing endocarditis disclosed by the detection of a murmur, but actual symptoms of cardiac weakness, such as precordial pain, dyspnœa, and orthnopœa. It is also notable that, although the onset with arthritic symptoms alone is commoner than any other one mode of onset, yet it existed in only 40 per cent. of all the cases. The number of cases in which the disease manifested itself at the onset by the occurrence of fever only, with or without slight and vague constitutional symptoms, is of special interest in connection with the practical question of diagnosis. It shows that rheumatism in children must be placed in that class of acute infectious diseases which do not necessarily show characteristic or localizing symptoms at the onset, and in which the diagnosis must be often for several days in doubt, until such symptoms have developed. When, as so often happens, the physician is confronted with a beginning acute infectious disease, in which the physical examination and history are not illuminating, he must not forget to number rheumatic fever among his possibilities. The onset with sore throat as the only symptom accompanying the fever, in 6 cases, is also of interest. In consideration of the fact that children only complain of sore throat in a small proportion of cases in which inflammation of the pharynx or tonsils is found on physical examination, it is very possible that the number of cases in which such inflammation was acutally a symptom of onset might have been larger if proper observation could have been employed. The interest of this inflammation as an initial symptom of rheumatic fever lies in the light which it may throw upon the question of the route of invasion of the infection.

The fever of rheumatism, while showing the widest variations in different cases, both as to the degree of temperature attained and as to its duration, nevertheless presents certain features which I believe are fairly characteristic of this disease. The amount of fever varies with the severity of the case and with the localization of the infectious process, every gradation being found from the most moderate fever curve, ranging between 99° and 100°, to one reaching 105° or even 106°. Hyperpyrexia, however, is rare in children, being very much less common than in adults; in an average case in a child the fever ranges between 101° and 103° at the height of the disease. The analysis of the cases which I made two years ago revealed that the temperature is higher in the cases with cardiac symptoms than in those with arthritic symptoms only, and, in general, higher in cases of pericarditis than in cases of endocarditis. The most notable characteristic of the temperature curve in rheumatic fever is the absence of any very wide daily variation in the temperature, the variation being, in the majority of cases, about one degree, and not often over two degrees; the morning temperature is almost always the lower. There are, of course, marked exceptions to this rule to be encountered at times. Nevertheless, sudden exacerbations, remissions, or critical falls in the temperature are uncommon. The temperature usually rises rapidly, but not with extreme suddenness, and in most of the arthritic cases and the milder cardiac cases falls rapidly, but not suddenly, to the normal. The maximum is by no means always reached near the beginning of the disease, as at any time a further exacerbation may occur, in which the fever increases fairly rapidly, but again not suddenly. The course of the fever may be prolonged over a period of weeks, with frequent exacerbations, especially in the cardiac cases, the longest fevers being found in cases of pericarditis. In such cases the termination is by a much less rapid lysis than in the arthritic and milder cardiac types. The duration of fever in 223 cases was as follows:

	Average.	Extreme.
In cases with arthritic symptoms only	4 days	1 day to 2 weeks.
In cases with endocarditis	12 "	1 " to 12 "
In cases with pericarditis	39 "	2 days to 12 "

In comparison with adults, the shorter duration of the fever in the articular cases, and the frequent exceedingly long duration of fever in the cardiac cases, form a very characteristic feature.

Constitutional symptoms, while sometimes present, are usually absent or slight in children. In some cases the fever of onset is accompanied by some headache and vague aching pains in the back and limbs, without pain on motion or tenderness in the joints. Such cases are frequently diagnosticated as grippe. There are almost never general symptoms referable to the nervous system. The profuse acid sweating, so commonly seen in adults, is compara-

tively uncommon in children, especially during the earlier years of childhood.

The joint symptoms of rheumatic fever in children deserve detailed mention. The first peculiarity of the disease which struck my attention in analyzing a series of cases, was the comparative infrequency of joint symptoms in children suffering from an acute infection which other evidences pointed to as being rheumatic fever. Out of 223 consecutive cases admitted to the Children's Hospital suffering from rheumatic fever, only 102 (45 per cent.) had joint symptoms. A second point is their great mildness in comparison with rheumatic fever in adults. Not only are the objective manifestations of swelling, redness, and heat comparatively infrequent, but the pain on motion is much less severe, often being so little as to cause only a slight limp, and the tenderness to pressure is often but slightly marked, or absent. Redness and swelling were present in only about 50 per cent. of the cases having joint symptoms, and in only 20 per cent. of all the cases of rheumatism in the series. The duration of the joint symptoms in children is also very brief, averaging a little less than two days.

This frequent great mildness and extremely short duration of the arthritic symptoms, combined, as they so often are, with very slight fever and no constitutional symptoms, account for the fact that rheumatism in children is so frequently overlooked. The symptoms, even when complained of by the child, are often attributed by the parent to "growing pains," and in other cases, in which the only manifestation is a slight limp in walking or complaint on using a limb, the disability is attributed to a strain.

As to the number of joints affected in children, there is wide variation. Only one joint may be affected, and the process is confined to one joint more often in children than in adults (10 per cent. of my series). Usually more than one joint is affected, but the number is in general fewer than in adults, a general polyarthritis being rare. The ankles are the joints most frequently affected, the knees being second and the wrists and hands third.

In this description of the arthritic manifestations of rheumatism I have laid stress on the general and typical peculiarities in children, but I do not wish to convey the impression that rheumatism never occurs in childhood in the same severe arthritic form as in adults. Cases are seen in which there is high fever, polyarthritis, with extreme pain on motion, tenderness, redness, and swelling, and in which there are even profuse acid sweats. Such cases are, however, not typical, and occur most often in older children. Indeed, a striking feature in the rheumatism of early life is the variation of its manifestations according to age. The younger the child, the more apt are the peculiarities mentioned above to be accentuated, arthritic manifestations being slight and cardiac manifestations prominent. The nearer adult life is approached, the more does the type of disease approach that seen in adults.

The cardiac manifestations of rheumatic fever in children are of importance, in the first place on account of their great frequency of occurrence. In my series, 91 per cent. of all cases had evidence of an organic lesion in the heart, which is a very much larger proportion than that observed in the rheumatism of adults. The commonest form is endocarditis, found in 91 per cent., pericarditis occurring in 26 per cent. It is, however, not so much this frequency of occurrence of cardiac involvement which constitutes the most important peculiarity of rheumatic fever in children, as it is the effect which acute endocarditis has upon the symptomatology of the disease. In the adult endocarditis is usually described as developing insidiously, betraying itself only by the appearance of a murmur. In children, endocarditis usually betrays itself by the occurrence of actual symptoms referable to the heart. Its occurrence may be insidious, as in the adult, but in very few of my cases did a murmur actually develop unaccompanied by cardiac symptoms. There were cases in which a murmur was found without symptoms, but in such cases it was not possible to know if acute endocarditis was actually present, or if the murmur was the relic of some previous attack. In 65 per cent. of my cases having a murmur there were actual cardiac symptoms. The symptoms themselves are the ordinary ones of cardiac weakness: dyspnœa, palpitation, and precordial pain in the milder cases, with the addition of œdema and cyanosis in the severer ones. It is not the character of these symptoms which is of importance, but their severity, obstinate duration, and danger to life. They are accompanied by fever and evidence of acute infection, and I believe the frequency of death from heart disease in children is not due to the overstraining of chronically damaged valves, but to the liability of children to acute rheumatic infection of the endocardium, causing cardiac weakness.

Another peculiarity of rheumatic fever in children lies in the occurrence of cases in which the only localization of the infection is the cardiac—the primary acute rheumatic endocarditis of childhood—cases which run a course characterized by fever and cardiac symptoms only.

The actual lesions show no important peculiarities, and are of comparatively little clinical significance. The mitral valve is very much the most frequently affected. Pericarditis in children shows no special peculiarities in symptomatology. The majority of cases go on to effusion.

One symptom of rheumatic fever, the occurrence of subcutaneous fibrous nodules, is mentioned by authorities as being more often seen in children. No case showing this symptom happened to occur in my series of 223 consecutive cases. I believe it to be rare, in Boston, at least, although its frequency may vary in different localities. It is probably more common in England.

Finally must be mentioned one of the most important peculiarities

of rheumatic fever in childhood, the liability to recurrence of the infection with varying manifestations. It is not uncommon for these recurrences completely to dominate childhood. There is no special order for the various clinical types to appear, and each attack may be mild or severe. At one time the symptoms are mainly cardiac, at other times mainly articular, at still other times a combination of both types. Any attack may be accompanied by pericarditis or followed, accompanied, or even preceded by chorea. In any attack the severity of the cardiac symptoms may lead to a fatal ending.

It is difficult to divide a disease with such varied and changing symptoms into distinct clinical types. The following division is based upon the character of the onset and the severity of the case; it must be remembered that at any time in an articular type of case cardiac symptoms may develop, and vice versa, and that even the mildest cases after recovery usually are left with a permanently damaged heart:

1. The mild arthritic type. These are cases with slight and brief fever and joint symptoms only, of a mild and brief, even fleeting, character, confined to one or few joints.

2. The severe arthritic type, occurring in older children, with a severe polyarthritis resembling the adult type.

3. Latent type. In these cases there is at the onset a period with fever as the only symptom. Later may occur cardiac or arthritic symptoms, which may be so mild as to escape notice, or which may at any time become severe.

4. Mild primary endocarditis. These cases are characterized by fever and slight dyspnœa, palpitation or precordial pain, the symptoms being of brief duration. Slight joint symptoms may develop in the course of the disease.

5. Severe primary endocarditis. These cases are characterized by fever and the whole train of the symptoms of cardiac incompetency. They usually run a prolonged and obstinate course, recovery occurring very gradually. At times death occurs from failure of the heart.

6. Mild pericarditis. In these cases precordial pain is the most prominent symptom, accompanied by fever and the other signs of cardiac weakness. They usually run a fairly long course. Effusion may or may not occur, and, a notable point, it may occur without increase in the severity of the cardiac symptoms. Cases of pericarditis almost invariably show endocardial murmurs also.

7. Severe pericarditis. In this type, the fever is high, long continued, and obstinate, and precordial pain is severe at the onset. Effusion always occurs. The symptoms of cardiac insufficiency become very marked, and their severity usually shows little relation to the amount of effusion, although at times a sudden increase in their severity occurs in parallel with a sudden increase in the extent of the effusion. Recovery is slow, and death, when it occurs, is due to cardiac weakness.

STATISTICS OF SEVENTY CASES OF GASTROSCOPY.

By Chevalier Jackson, M.D.,

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The superiority of inspection to deduction as a process in diagnosis requires no argument. It may be measured by the usefulness of the cystoscope. The stomach is more amenable to endoscopic methods than is the bladder, and its pathological importance is vastly greater. Curiously enough, it was the success of the Nitze-Leiter cystoscope that retarded gastroscopy. The earnest and ingenious efforts of von Mikulicz, Rosenheim, and Rewidzof in this field were ever fruitless, because they started with the radical and radically wrong principle of obtaining a view through a lateral window by an arrangement of lenses and prisms. Such an optical apparatus necessarily involves a fatal defect—a blind tip to an instrument which is passed in the dark by the sense of touch alone. Such passage of a straight rigid instrument into the stomach is not only exceedingly difficult (von Mikulicz considered it impossible in all cases, Rosenheim in many), but is dangerous.

There are several differences in anatomical conditions, not necessary to enumerate, that render the cystoscopic principle unsatisfactory here. It is sufficient to allude to the much greater size of the stomach. When one dilates the stomach he pushes its walls far away from the reach of the tube; walls which otherwise would collapse over the tube mouth, to be examined and palpated by the probe and tube. The portion of the stomach nearest the centre line of the body is the most easily examined. The collapsed stomach is relatively small, and much of it is near the middle line (Fig. 1). When one distends the stomach he pushes most of the otherwise explorable area away from the central line and thus laterally out of range. The diaphragm is rendered much less movable when the stomach is distended, and, furthermore, thus is rendered impossible the practice of a most valuable part of the technique, namely, the manipulation of the abdomen externally by an assistant, which brings into view the fundal and pyloric ends. A lens system and an inflated stomach prevent sponging away of secretions with which many lesions are covered.

The position and shape of the stomach in the living subject has been most curiously misunderstood. Fig. 2 is traced from one of the classical text-books on anatomy. Whatever may be the position and shape of the stomach in the cadaver or in the living subject after the abdomen is opened, it was certainly not in any such position in any of the seventy cases examined gastroscopically by me. My method of outlining the stomach is to find a given boundary with the extrem-

ity of the tube. The distal end of the tube is felt by the abdominal palpator, who makes a mark on the patient's skin with a skin

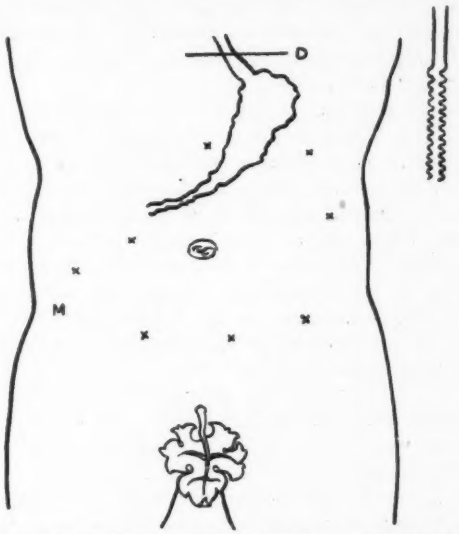

FIG. 1.—Position of the stomach in the case of Isabel A. Crosses show where the wall of the stomach was intentionally pushed by the gastroscope. The schema in the upper right-hand corner shows the other plane of the stomach.

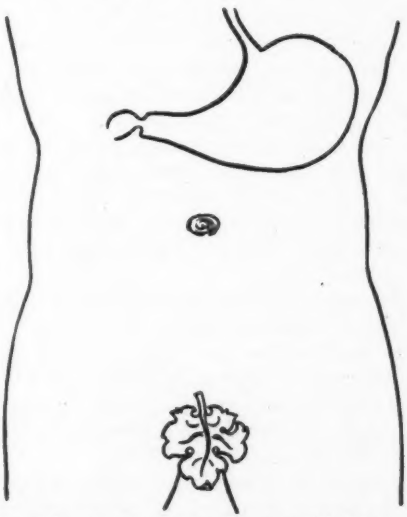

FIG. 2.—Position of the stomach as shown in a classical text-book on anatomy.

pencil. Another position on the boundary is then found and marked, and thus a series of marks dot the skin of the abdomen corresponding to the stomach outlines. An obvious source of error is the drag of the tube, which may displace the stomach. This can be avoided by a careful watch through the tube and care to make a vertical insertion for each mark.

The stomach wall can be pushed into almost any position, as shown in Fig. 1. The position of the stomach in the case of Isabel A. is shown here. It gave the impression of a loose bag dangling on the end of the gastroscope, freely movable in all directions, by either the movement of the gastroscope or the manipulations of Dr. Harold A. Miller, who was palpating the abdomen externally. The diagram at the upper right hand corner of the illustration shows schematically the other plane as it appeared to be when gastroscopically examined. Passing down the œsophagus, as soon as one passes the cardia folds and wrinkles are encountered, a slight deflection bringing either the anterior or the posterior wall into view.

The degree of motion shown in Fig. 1 is obtainable only under the relaxation of deep anesthesia. When gastroscopy is attempted, under morphine narcosis, as Mikulicz attempted it, the musculature of the diaphragm pulls upon the central tendon, so that the gastroscope is guyed rigidly like a tent pole, and if the stomach can be entered at all, only such portion can be inspected as lies in a line with the axis of the entry of the tube. When relaxed under deep anesthesia the hiatus œsophageus does not relax or enlarge so as to permit of motion; but the entire dome of the diaphragm can be moved sidewise because it is a dome. If it were a tightly stretched membrane, there would be no yield in any direction; but being arched, its "slack," as one might say, permits of a range of motion of from 10 to 15 cm., provided the central tendon is not pulled upon from all sides by the diaphragmatic musculature.

In regard to the safety of gastroscopy, my statistics show that in no instance has harm followed. Occasionally, stiffness or soreness in the neck has been noted for one or two days. There has been no other unpleasant symptom in the whole series of 70 cases, and no patient has died, from any cause whatsoever, within thirty days after the gastroscopy.

The utility of gastroscopy in the removal of foreign bodies is obvious. Its highest function, however, is as an instrument of diagnosis in the hands of the gastrologist, and of the gastroscopic assistant of the abdominal surgeon, who will assist the surgeon not only in diagnosis, but in the operating room during the operation, by working through the mouth in conjunction with the surgeon whose hand is in the abdomen.

In my gastroscopic work I have had the following positive findings in 18 patients out of the 70 examined, some patients having more than one condition:

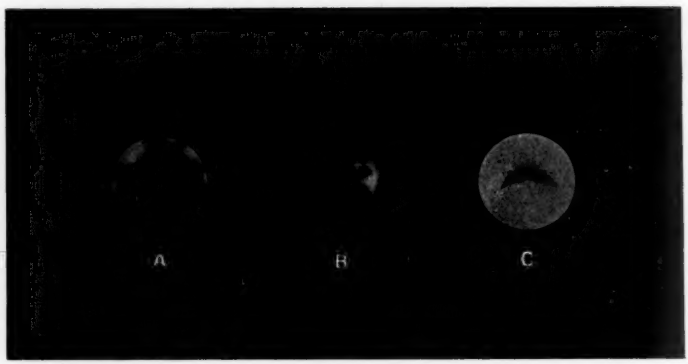

FIG. 3.—*A*, gastroscopic view of a gastrojejunostomy opening drawn patulous by the tube mouth (patient of Dr. George L. Hays). *B*, carcinoma of the lesser curvature (patient of Dr. Joseph H. Barach, afterward surgically explored by Dr. John J. Buchanan). *C*, healed perforated ulcer (patient of Dr. John W. Boyce).

	Cases.
Chronic gastritis	6
Gastroptosis	3
Gastrectasia	2
Malignant disease, cardia	3
Malignant disease, pylorus	3
Malignant disease, lesser curvature. (Specimen taken in two instances)	3
Peptic ulcer	8
Peptic ulcer cured(?)	1
Negative results of value	1
Foreign body removed	1
Gastric syphilis	1

Two cases may be cited as illustrating the possibilities of diagnosis by this method:

CASE XXV.—Isaac L., aged fifty-seven years, was referred for gastroscopy by Dr. Joseph H. Barach. For two years the patient had difficulty in swallowing. On admission he could swallow bread, but not liquids; coffee went down better than water. He had lost 13 kilograms in six months. The area of gastric tympany seemed normal, and there was no palpable mass. The patient never vomited, and the stomach tube was arrested 40 cm. from the upper teeth, so that it was impossible to examine the stomach contents. The patient was admitted to the Eye and Ear Hospital, for gastroscopy. Under the relaxation of deep general anesthesia no mass was palpable. The gastroscope was readily introduced through the cardia. His dysphagia was thus demonstrated to be due to cardiospasm, which was also visible to the eye when the patient came partly out of the anesthesia. As soon as the tube mouth reached the lesser curvature in the middle third of the stomach, I pronounced the case one of carcinoma. The appearance was characteristic and unmistakable. A mass was seen, dark gray in color, occupying a breadth equal to four rugæ. Instead of the usual pliant infolding of the normal membrane as seen through the gastroscope, the large grayish mass shown at the right in *B*, in Fig. 3, flopped in front of the tube in a way that contrasted with the size, form, and motion of the normal membrane even more than did the color.

After the examination the patient was able to swallow. He gained in weight and strength so rapidly that one of his physicians, a thoroughly competent man, disputed the diagnosis of malignancy. Pain after eating was still present, however, and the patient consented to laparatomy, which was done by Dr. John J. Buchanan. He found an inoperable carcinoma of the lesser curvature.

This was a case which, aside from œsophageal spasm, presented clinically only the functional dyspepsia which often precedes the development of the characteristic symptoms of malignancy. Yet the carcinoma, unrecognizable except by the gastroscope, had already developed beyond the hope of operative cure.

CASE XXIV.—J. G., an epileptic of rather limited intelligence, was admitted to the Western Pennsylvania Hospital, complaining of burning pain in the stomach, radiating to the right nipple. Pain

was aggravated by eating, but was present at times when the stomach was empty. The patient had lost much weight. He had vomited once only, the ejected material containing a little blood.

Gastroscopy was done under general anesthesia. The mucosa showed evidence of chronic gastritis, and was covered with thick, pasty, mucopurulent secretion. A considerable quantity of fluid pus of yellowish color poured into the gastroscope, evidently coming from the left of the cardia. Smears by Dr. W. H. Allen showed numerous bacteria, most prominent being a Gram-positive organism, probably Streptococcus pyogenes, and a Gram-negative diplococcus resembling Micrococcus catarrhalis. No opening through the mucous membrane could be found, but the pus streamed from the left into the gastroscope in a way that precluded the idea of its being a mere exudate from the mucosa. More than 100 c.c. was drawn off through the drainage canal of the gastroscope. The diagnosis, therefore, was perigastric abscess, with perforation.

More careful inquiry into the previous history showed that the patient had suffered four years previously from a severe febrile illness of two week's duration, which he reported as typhoid fever. He remembered that during this period he had suffered from a severe pain in the left hypochondrium, which compelled him to lie curled up in bed bending to the left side. His physician had told him that his "liver was enlarged." When he came to the hospital the left lobe of the liver seemed quite tender, but there was no enlargement. Whether his original abscess was subdiaphragmatic or located in the liver, I am unable to say. After gastroscopy his symptoms improved so that he refused further treatment, and soon drifted away from the city.

Case LXIX.—Daniel B., aged sixty-two years, was admitted to the Western Pennsylvania Hospital to the service of Dr. John W. Boyce, complaining of feeling weak, and of loss of appetite, emaciation, and headache, all these symptoms appearing gradually about one year previously. There was no nausea or vomiting. The temperature was normal, the pulse 96, and the respirations 24.

After the mouth of the gastroscope passed the cardia, in the first 4 cm. of the passage, the anterior and posterior walls of the stomach opened up in normal folds ahead of the tube mouth. Below this, however, the tube mouth entered a cavity with smooth unwrinkled walls. At the bottom of this cavity was a crescentic slit-like depression looking somewhat like the primiparous os uteri (*C*, Fig. 3). When the mucosa was sponged clean of bubbly secretion, the slit-like depression was found to have a considerable depth, which, however, was not probed, although to have done so would have been technically easy and probably harmless. The surrounding mucosa was of pale pink color, without rugæ, and when the tube mouth was withdrawn the depression was still visible in the same position in a cavity, the wall of the stomach at this point being evidently held open by adhesions to the abdominal parietes or viscera. Fully

to appreciate the picture, it is necessary to realize that, to paraphrase, "nature abhors a cavity." The empty stomach is collapsed. When examined gastroscopically, it opens up ahead of the tube and collapses after its withdrawal, in a manner similar to that of the vagina upon the introduction and withdrawal of the vaginal speculum when used upon a patient in the dorsal position. But here was a case in which the anterior wall of the stomach was adherent, so that it was held up (dorsal decubitus); thus the posterior wall dropped away by gravity and left a cavity. The cicatrices and adhesions were such that no mucosal folds could be produced in the neighborhood of the slit. When Dr. Ogilvie tapped upon the abdomen the vibrations were seen to be beautifully transmitted in waves over the upper (anterior) wall of the stomach.

The picture was beautifully clear, and my colleagues, who saw for themselves, and I felt justified in pronouncing the lesion the cicatrix of a healed perforating gastric ulcer. Had it been an operable lesion, it could have been precisely located by holding the tube mouth against it while the abdominal surgeon cut through the stomach wall from the celiotomic wound.

CASE XX.—Margaret Z., a servant at the Western Pennsylvania Hospital. Six months before, gastrojejunostomy had been performed by Dr. George L. Hays. Complete symptomatic cure had followed. Under general anesthesia I passed the gastroscope, and readily found the anastamotic opening in the form of a slit, which, when pulled open by the instrument, showed a slightly puckered border, below which could be seen the mucosa of the jejunum (*A*, Fig. 3). At a point about 4 cm. from the opening was a shallow ulcer.

Apart from the ease of examination, this case is interesting in showing the existence of an ulcer, in spite of perfect symptomatic cure. Postoperative study of ulcer cases will perhaps show interesting results. Dr. Braasch, of the Mayo clinic, wrote me (September, 1907) that he has observed a Murphy button in situ after operation. Dr. W. L. Rodman, with my assistance, examined gastroscopically a gastrojejunostomy wound two weeks after operation, and saw the opening and the non-absorbable sutures in situ.

Gastrology is perhaps the most satisfactory as to diagnosis of all departments of internal medicine. But this satisfaction is relative, not absolute. Psychic affections sometimes so closely simulate ulcer (even to the apparent vomiting of blood) that the distinction is only made by what seems an intuition that belongs rather to the art than the science of medicine. Carcinoma of the stomach is rarely recognized in time for curative operation. Syphilis of the stomach is well known pathologically, but in so complete a work as *Nothnagel's System*, one leaf seems enough to cover our knowledge of its clinical history. There is, then, in this department alone room for direct inspection to work as great a revolution as did physical methods in thoracic disease.

MYELOGENOUS LEUKEMIA AND ITS TREATMENT WITH X-RAYS.

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In the last two years there have been 4 cases of myelogenous leukemia observed at the Cooper Medical Clinic, and to these I am adding one seen in private practice. In this same period at the clinic we have seen 1 case of Hodgkin's disease, 1 of Banti's disease, no cases of lymphatic leukemia, no cases of pernicious anemia, and 1 case of anemia with enlarged spleen of doubtful nature. Of some 700 purely medical cases seen yearly at the above clinic, there are 2 or 3 of myelogenous leukemia. As to the etiology, there is nothing positive to say. Two of the patients resided for many years in the malarial districts of California, and still denied ever having had malaria. In only one case was there a history of this infection. Nor were there any other features to which etiological importance could be attached. A history of syphilis was not obtained in any of our patients. All were men varying in age from forty to sixty-two years. Their antecedents and offspring had not suffered from any similar disease. On purely clinical grounds a certain incomplete resemblance to malignant disease was thus seen; this has been commented upon by other observers. The resemblance is further borne out by the course of the disease, its reaction to the *x*-rays, and its occasional improvement by intercurrent infection.

Opinions are divided as to the pathology of the leukemias and Hodgkin's disease. Some regard the processes as essentially hyperplasia, the increased number of leukocytes in the blood, due, in the case of lymphatic leukemia, to the passive washing out of lymphocytes into the blood current, and in myelogenous leukemia to the active migration of various forms of leukocytes following chemotactic influence. More recently these diseases have been regarded as malignant. Atypical growths of cells and a tendency to invade the capsules of the lymph glands and adjacent tissues are the indications of this malignant nature. According to Banti,[1] all leukemias and Hodgkin's disease are really neoplastic. The anatomical changes in the bone marrow, lymph glands, and spleen, where large groups of new cells invade neighboring tissues, are those of sarcomatosis. The walls of the bloodvessels, especially the veins, and even the surrounding bone, are invaded, and the changes in the blood are simply due to the tumor cells gaining entrance to the circulation. If such invaded vessels be thrombosed, the number of leukocytes will drop. In pseudoleukemia such sarcomatous

cells have not entered the circulation. When they do, the case becomes clinically one of lymphatic leukemia. Banti regards as true metastases the enormous growths of myeloid or lymphatic tissue in other viscera. Myelogenous leukemia, according to this view, is a systemic myeloid sarcomatosis of the blood and lymph-forming apparatus, while lymphatic leukemia is a systemic lymphadenoid sarcomatosis of these organs. A similar view of malignancy concerning Hodgkin's disease, largely based on anatomical findings, has been expressed by Walter Gibbons.[3]

Clinically, leukemia corresponds closely to malignant neoplastic conditions, and the following clinical factors support the view that it is really a malignant disease: Its onset is nsidious. It is practically always fatal. The cachexia of leukemia occurs in the practical absence of fever. It is occasionally benefited by intercurrent affections, a feature true even of such an eminently malignant disease as cancer of the breast. It is usually favorably influenced by the *x*-rays. Objections based on the rare occurrence of spontaneous recovery in the disease are not very forceful, since similar instances of spontaneous recovery have been observed in undoubted cases of malignancy (Czerny),[3] as, for instance, after incomplete operation, or after intercurrent infection in cancer of the breast. That, in a few recorded cases, a marked hereditary tendency has been noted in leukemia does not harmonize with the usual picture of malignancy. As for the acute leukemias—aside from the blood findings, they do not resemble closely the chronic forms.

Pathologically, the large accumulations of new cells found in the tissues in myelogenous and lymphatic leukemia, pseudoleukemia (Hodgkin's), and chloroma have been differently interpreted. Some pathologists have regarded the picture as essentially that of hyperplasia (Lazarus,[4] Pinkus[5]) occurring in the bone-marrow, lymph glands, and spleen. Others—and I have already cited Banti—believe that the new growths are malignant. In favor of the latter view in myelogenous leukemia is the finding in many organs (lung and liver, for instance) of masses of cells identical with those of bone-marrow, foreign to the particular organs involved, apparently metastases. Growth of this new tissue may also be by direct extension.

In chloroma—green cancer of d'Aran—in which the blood is identical with lymphatic leukemia, this invasive character is particularly marked. Collections of new cells of a greenish color form in and between the bones, especially of the skull and face. By compression blindness, deafness, and various paralyses result. These greenish tumors also occur scattered through the different organs. Stengel[6] calls the disease sarcomatous.

[3] Amer. Jour. Med. Sci., Nov. 1906.
[4] Nothnagel's System, 1901.
[5] Zeitschrift f. Krebsforshung, 1907.
[6] Text-book of Pathology, 1903.
[5] Ibid.

In lymphatic leukemia the question, too, is how to interpret the new formations. Are they lymphomas of hyperplasia, or are they malignant and sarcomatous?

In another class of cases, however, the combination of leukemia and anemia, called by Leube leukanemia, the resemblance to an acute infection or acute toxemia of rapid onset is striking. Such a case, in which the blood resembled that of a severe leukemia, ending in three and a half months with recovery, was recently described by Teeter[7]. Emerson,[8] in describing 3 cases of acute leukemia, points out the resemblance clinically to an acute infection of the bone-marrow.

Symptoms. These are insidious. One man of our series kept at work seven months after first noticing any disturbance. The first symptoms noted by the patients were dull pains in the left abdomen, and those of anemia, such as weakness, dyspnœa, and pallor. A tumor in the left abdomen was noticed by two of the men. More particularly for the relief of the pain did they go to the physician; this was not severe, so that the first visit was postponed for weeks or months. In 1 case there was also substernal pain. The anemic symptoms were also insidious in onset and had no special characters. In 2 cases there was vomiting and in 2 cases epistaxis. Ringing in the ears and dizziness were the only nervous symptoms noted, and these could also be attributed to anemia. In 3 cases the appetite remained good. In the others it was lessened. The bowels were regular. A loss of weight was usually noted, although it was not excessive. In 1 case this amounted to 19 pounds in two years. One patient was still well nourished. With the progress of the disease the emaciation increased. The skin looked pale, in 1 case it was jaundiced. Arsenical pigmentation and evidence of slight *x*-ray dermatitis about the splenic region were observed, due to treatment. Slight œdema at the ankles was found in all cases. The pulse, often rapid, was not distinctive. The temperature was elevated at some time in all the cases.

The lungs were found uninvolved in 3 cases. In the fourth case an old tuberculous lesion was overlooked. The fifth case has now a lesion of the right apex, doubtlessly tuberculous. Enlargement of the cardiac dulness transversely occurred in 4 cases. The largest heart extended from the right sternal border to 4 cm. to the left of the left mammary line. A faint systolic murmur was noted occasionally at the apex or base, but in no case was a diagnosis of endocarditis made. Enlargement of hepatic dulness was present in all cases, though the edge of the organ was not always felt. In one case the vertical measurement in the right mammary line was 22 cm. When the liver edge was felt, it was found firm, smooth, and rather

[7] Jour. Amer. Med. Assoc., February 16, 1907.
[8] Johns Hopkins Hosp. Bull., March, 1907.

rounded. The spleen was enlarged in all cases. In 3 it was enormous, filling out nearly the entire left side of the abdomen, and in these and in a fourth case the splenic tumor was easily seen on inspection of the abdomen. The largest splenic dulness measured 32 x 30 cm., reaching to 6 cm. below the umbilicus and 2.5 cm. to the right of the median line. The organ felt firm, smooth, its edge somewhat rounded and notched more than normally. It was not painful to palpation. The size of the spleen in a general way varied directly with the condition of the patient and with the number of leukocytes, diminishing in size as the patient convalesced and as the leukocytes diminished. Comparing different patients, however, no such parallelism occurred; for instance, in Case I, at a time when there were 632,000 leukocytes per c.mm., the spleen reached at expiration only 4 cm. below the costal margin; while in Case V, with 48,000 leukocytes, the organ extended 6 cm. below the umbilicus. Both of these cases were seen about six months after the first symptoms had appeared. There was slight general enlargement of the lymph glands in Case IV, possibly due to the tuberculous infection subsequently found. In Case III the lymph glands were somewhat enlarged, but not tender. The nervous system and genitalia were negative. The urine in 3 cases showed at first only a trace of albumin, with hyaline and granular casts in 2 cases.

Blood. In this series of cases the average number of leukocytes when first seen was 312,000; that of the red cells was 2,860,000. The relation of white to red cells was that of 1 to 9. The average hemoglobin content was 50 per cent. When first seen the myelocytes constituted from 30 to 64 per cent. of all the leukocytes. Under the *x*-rays their numbers were both actually and relatively reduced. Through the influence of an acute miliary tuberculosis in one case they disappeared entirely. The leukocytes in this case dropped from 300,000 to 6000 in five weeks.

This brings up the interesting point of the influence of complicating infections upon leukemia, a subject extensively considered by Dock.[9] From the literature and from his cases, it is shown that, under the influence of complicating infections in leukemic patients, there is a disposition for the leukemic characters of the blood to disappear more or less completely, the spleen, liver, and lymph glands often growing smaller. In the majority of cases the leukocytes are diminished. In about half the cases the number is reduced to normal or less than normal. These conditions hold true for acute infections, in which the number of leukocytes may rapidly diminish within such a short period even as three to ten days. In one of our cases the eosinophilia were absent in several examinations, a feature also noted by Simon. Joachim[10] has recently described 2 cases of what he calls

[9] Amer. Jour. Med. Sci., April, 1904.
[10] Deutsch. Archiv f. klin. Med., lxxxviii.

"mast-cell leukemia." These mast cells constituted over 50 per cent. of the leukocytes present. It is a variety of myelemia with a prognosis less favorable than the ordinary from. The blood findings in our 5 cases are epitomized in Chart I.

CHART I.—THE BLOOD IN FIVE CASES.

Date.	Red cells.	Hemoglobin.	Leukocytes.	Small mononuclears.	Large mononuclears.	Polynuclears.	Eosinophiles.	Myelocytes.	Basophiles.	Nucleated reds to 500 leukocytes.	Remarks.
Case I.											
Aug. 23, 1905.	2,000,000	30–35	632,000								
Jan. 23, 1906.	3,120,000	60.0	3,400	20.0	10.0	66.7	0.0	3.3	0.0	0.0	Patient doing well.
Apr. 9, 1907.	3,464,000	60.0	85.260	4.6	2.8	69.7	1.8	21.0	0.0	2.0	
Case II.											
Mar. 24, 1905.	4,000,000	67.0	172,000	13.0	10.0	37.0	2.0	31.0	3.0	0.0	Death Sept., 1906
Case III.											
May. 31, 1905.	2,500,000	42.0	570,000	3.0	1.0	30.0	2.0	64.0			
Aug. 21, 1905.	3,650,000	70.0	83,600	4.0	7.5	63.1	1.0	23.9	0.5	7.0	Death Oct. 29, '05
Case IV.											
Nov. 22, 1905.	2,750,000	53.0	160,000	2.5	0.5	40.0	1.5	55.0	1.0	7.0	Miliary tuberculosis
Dec. 26, 1905.	2,220,000	38.0	3,600	4.0	8.0	88.0	0.0	0.0	0.0		Death Jan. 1, '06
Case V.											
Mar. 11, 1907.	3,050,000	55.0	72,000	5.0	3.3	51.7	2.0	30.4	7.6	4.0	Left the wards
Apr. 6, 1907.	3,950,000	60.0	63,600	0.6	1.9	53.3	6.7	37.0	0.6	2 normoblasts.	

DIAGNOSIS. In these cases with a typical blood picture and enlarged spleen the diagnosis was easy. Mixed forms, however, do occur, such as those seemingly combined with pernicious anemia. In lymphatic leukemia the diagnosis is evident by the increase of lymphocytes; myelocytes are not found. In cases of so-called aplastic leukemia, in which the clinical picture of leukemia is present, but with no increase of leukocytes, the diagnosis is based upon the changed blood formula. In an anomalous case studied by Ewald,[11] lasting seven weeks, autopsy findings were identical with myelogenous leukemia, and still the blood gave no sign of it during life.

COURSE AND PROGNOSIS. The prognosis is bad. The disease, in most cases, progresses in spite of treatment and terminal infectious or terminal hemorrhages are likely. Still, with the palliative action of the *x*-rays the prognosis would seem to be better than formerly. This bad prognosis is exemplified in our 5 cases. In Case III death occurred with anasarca and subdural hemorrhage two and

[11] Berliner klin. Woch., xlii, Nr. 26.

one-half years after the onset of the illness. In Case IV death resulted from miliary tuberculosis nine months after the first symptoms of leukemia. In Case II death ensued two and one-half years after the onset of the first symptoms. Case V has been seen only recently. The patient has been sick for six months, and has at present a catarrh of the right apex, no doubt tuberculous. Case I is remarkable, and will be spoken of especially under treatment. With occasional applications of the *x*-rays for eight months, at the end of a year he had practically recovered. It is now over two years since his symptoms were first noted, and in spite of the fact that he was sixty-two years of age, had a distinctly dilated heart and a good trace of albumin in his urine, with 632,000 leukocytes per c.mm., and with the hemoglobin at 30 to 35 per cent., he is now practically well, able to handle his business interests, and is otherwise strong. The blood picture, however, is far from normal.

TREATMENT. As in other severe chronic conditions, it is imperative that the patient be well fed and receive considerable rest. Our clinic patients were put to bed. They received the regular ward diet, with laxatives as required. Under this careful *regime*, with applications of *x*-rays, they were able to leave the ward, except in one instance, in a few weeks and return to light work. They then received *x*-ray applications once or twice weekly as ambulatory patients. Eventually, as their conditions grew worse, they returned to the ward, with the results previously given. Owing to gastro-intestinal irritation, arsenic, either as Fowler's solution or Asiatic pill, was found unsatisfactory. Hypodermic use of arsenic was not tried. By far the most satisfactory results were obtained by giving *x*-rays in small and repeated doses. Except toward the approach of death, the patients grew stronger and the spleen and liver were reduced in size. The leukocytes, especially the myelocytes, were quantitatively reduced. The red cells and the hemoglobin were more difficult to influence. The latter could not be raised to normal; and, even in our most favorable cases, myelocytes were still found.

The method of application of the *x*-rays can be best illustrated by following Case I. Applications were made three times weekly for three months, then every five days for two months, and as the blood picture improved and the patient improved the treatment was given at longer intervals. The current was obtained from a coil; a medium high or medium tube was generally used, occasionally a medium soft; the distance of the tube from the body was 25 to 30 cm; time of exposure, seven to fifteen minutes; amperage, 7 to 10 with the higher tubes; voltage, 10 to 120. Interruptions were made with a Wehnault or mechanical interrupter (2600 to 3000). The regions exposed were the splenic, anteriorly and posteriorly, the dorsal, the two thighs, the epigastric, and the sternal. Dermatitis was carefully avoided. The *x*-ray applications were

started three weeks after the patient was first seen. During these three weeks while taking arsenic and iron, he had grown appreciably worse, but with the beginning of the *x*-ray treatment the improvement began and continued as long as the *x*-rays were given. This improvement was felt by the patient himself, was found on physical examination, and was particularly striking in the changed blood picture. At the beginning of treatment the blood was as follows: red cells, 2,000,000; white cells, 632,000; hemoglobin, 30 to 35 per cent. Three and a half months afterward the reds were 3,784,000; whites, 8400; hemoglobin, 50 per cent. In September, 1906, five months after the cessation of *x*-ray applications, the reds were 4,000,000; whites, 10,000; hemoglobin, 70 per cent. A palliative though far less successful effect was shown in three other patients. The number of treatments given monthly with the changed blood picture are tabulated in Chart II. Case IV was complicated by miliary tuberculosis, so that the results were obscured; and in Case V also we believe pulmonary tuberculosis to exist. In Case I the result, as stated, was striking. It was not necessary to place this patient in bed. He lived at home and came to the Lane Hospital for his *x*-ray applications. Possibly the better result in this case was due partly to the fact that the patient was better able to care for himself, the other 4 being clinic and dispensary patients.[12]

The palliative action of the *x*-rays in leukemias and pseudoleukemias is now generally conceded. Indicating the profession's interest, Shirmer[13] was able to collect 109 articles on the subject published in two years. We have had no experience with the *x*-rays in lymphatic leukemia, but in 1 case of Hodgkin's disease, of four years' duration, the general condition of the patient has been much improved and the affected lymph glands made considerably smaller by the *x*-rays. Most of the good results, however, have occurred in cases of myelogenous leukemia. Cheney[14] has reported one such case, seen at the Lane Hospital. It must be understood distinctly, however, that in the leukemic conditions the *x*-rays are not curative. They are at most palliative; relapses occur under this as under other methods of treatment, but for several months—over twelve in one of our cases—the patient may be free of symptoms. In a small number of cases, approximately 10 per cent., the treatment seems valueless, or even harmful. The action on the leukocytes may be rapid. After a preliminary rise within a few days or in a few hours there may be a diminution of many thousands. The mononuclears are particularly affected. The red cells and hemoglobin may or may not be increased, but a more definite achievement is the improvement of the patient, as shown by increased appetite, increased

[12] I acknowledge with thanks the kindness of Dr. Oliver, of the Cooper Pathologica Department, and of Dr. Lehman, of the Cooper Dermatological Department, in furnishing some of these data.

[13] Central. f. d. Grens. d. Med. u. Chir., ix, 15.

[14] Medical News, 1904

CHART II.—RECORDS OF BLOOD COUNTS WHILE PATIENTS WERE UNDER TREATMENT.

Date.	X-ray applications during the month.	Red cells.	White cells.	Hemoglobin.	Remarks.
Case I.				Per ct.	
Aug. 23, 1905.	3	2,000,000	632,000	30–35	
Aug. 29, 1905.		2,240,000	528,800	40	
Sept. 6, 1905.	13	2,500,000	255,200	40	
Sept. 16, 1905.		2,360,000	245,600	38	
Oct. 4, 1905.	13	3,000,800	360,800	50	
Oct. 24, 1905.		3,160,000	133,600	52	
Nov. 2, 1905.	6	3,550,000	62,400	55	
Nov. 28, 1905.		3,620,000	13,200	60	
Dec. 16, 1905.	6	3,784,000	8,400	60	
Jan. 4, 1906.	6	3,968,000	16,800		Laryngitis of 10 days' duration.
Jan. 28, 1906.		3,120,000	3,400	60	
Feb. 14, 1906.	4	3,440,000	3,000	60	
Mar. 10, 1906.	4	2,820,000	4,600	60	
Apr. 3, 1906.	4	2,400,000	3,600	50	Discontinued x-rays after earthquake of 1906.
Sept. 18, 1906.		4,000,000	10,000	70	
Apr. 9, 1907.		3,464,000	85,260	60	
May. 10, 1907.	2	3,536,000	109,100	70	
June 14, 1907.	4	4,100,000	104,800	67	
July 24, 1907.	4	4,100,000	165,000	67	
Case II.					
Apr. 7, 1905.	4	3,960,000	146,000	67	Has been receiving x-rays
Apr. 28, 1905.		4,000,000	100,000	75	elsewhere; now ambu-
May 3, 1905.	13	4,000,000	97,000	75	latory.
May 24, 1905.		3,750,000	116,000	72	
June 2, 1905.	13	4,000,000	140,000	78	
June 30, 1905.		3,600,000	115,000	74	
July 11, 1905.	13	3,900,000	70,400	76	
July 28, 1905.		4,000,000	54,000	80	
Aug. 16, 1905.	13	4,050,000	41,600	76	
Aug. 24, 1905.		4,000,000	80,000	80	
Sept. 1, 1905.	9	4,250,000	40,000	80	
Sept. 27, 1905.		4,650,000	60,000	82	
Oct. 5, 1905.	13	4,650,000	79,000	80	
Oct. 25, 1905.		4,650,000	56,000	80	
Nov. 9, 1905.	13	3,500,000	54,000	76	Entered Lane Hospital
Nov. 22, 1905.		3,900,000	64,000	75	Nov. 30.
Dec. 7, 1905.	2	3,750,000	40,000	74	Left hospital Dec. 14.
Dec. 27, 1905.		4,000,000	70,400	80	
Jan. 10, 1906.	4	3,500,000	80,000	68	Ambulatory; reëntered
Jan. 31, 1906.		3,250,000	90,000	68	Lane Hospital Jan. 19.
Feb. 12, 1906.	10	3,500,000	104,000	67	Left hospital Feb. 5, re-
Feb. 20, 1906.		3,500,000	112,000	65	entered Feb. 15.
Mar. 5, 1906.	13	3,500,000	160,000	63	Left hospital April 16.
Mar. 14, 1906.		3,000,000	160,000	54	X-ray treatment then
Apr. 2, 1906.	7	3,000,000	120,000	55	discontinued.
Apr. 10, 1906.		3,350,000	78,000	64	Death in September.
Case III.					
May 15, 1905.	8	3,150,000	456,000		
May 31, 1905.		2,250,000	510,000	42	
June 12, 1905.	13	2,600,000	460,800	50	Entered Lane Hospital
June 30, 1905.		2,100,000	563,000	42	June 28, 1905
July 11, 1905.	19	2,500,000	540,000	45	
July 19, 1905.		3,000,000	320,000	54	
July 26, 1905.		3,250,000	180,000	60	
Aug. 2, 1905.	15	3,350,000	187,600	62	Left hospital Aug. 16.
Aug. 16, 1905.		3,650,000	104,000	70	
Aug. 30, 1905.		3,500,000	80,000	70	
Sept. 5, 1905.	13	3,650,000	70,000	70	Ambulatory.
Sept. 27, 1905.		3,350,000	136,000	64	
Oct. 12, 1905.	11	2,800,000	260,000	50	Reëntered hospital Oct.
Oct. 25, 1905.		2,600,000	208,000	50	16; death, Oct. 28, '05.
Case IV.					
Nov. 14, 1905.	3	2,750,000	300,000	50	Entered hospital Nov.
Nov. 22, 1905.		2,750,000	160,000	53	21, 1905.
Nov. 29, 1905.		3,950,000	160,000	77	
Dec. 7, 1905.	9	2,750,000	40,000	52	Death, Jan. 1, 1906; mi-
Dec. 21, 1905.		1,850,000	6,000	36	liary tuberculosis.
Dec. 28, 1905.		2,220,000	3,600	38	
Case V.					
Mar. 11, 1907.	4	3,050,000	72,000	55	Entered Lane Hospital,
Mar. 22, 1907.		3,100,000	64,000	55	March 19; x-rays given
Mar. 30, 1907.		3,450,000	45,500	60	first, March 20.
Apr. 6, 1907.	3	3,900,000	63,600	60	Left hospital, April 6.

strength and weight, and in women by the reappearance of menstruation. The spleen and liver usually grow smaller.

As to the mode of action of the *x*-rays, we know that a leukolytic body or bodies develop under treatment. Helber and Linser[15] have shown that serum from an animal treated with *x*-rays, when injected into another animal, causes a leukopenia. Control serum caused a leukocytosis. The extract of the still living, though extirpated, spleen, which organ had been exposed for several hours to the *x*-rays, caused a reduction of leukocytes upon being injected into the normal animals. The extract of a control spleen not exposed to the *x*-rays produced a leukocytosis (Milchner and Wolf[16]). The most convincing work along this line has been done in this country by Capps and Smith.[17] It was proved by them that the serum of leukemic patients improving under *x*-ray treatment causes a leukopenia when injected into animals; added to a hanging drop of leukocytes from another individual, a disintegration of cells, especially the mononuclears, occurs. They also injected such serum into another leukemic patient not under *x*-ray treatment and succeeded in demonstrating a rapid and decided drop of leukocytes in the injected patient. With repeated injections, however, a partial immunity to the injected serum was established. Whether this leukolytic substance or substances prevents the excessive formation of leukocytes in the blood and lymph-forming organs, or dissolves the leukocytes after they have been formed, is not known. Nor have the studies of metabolism, especially concerning the uric acid output of patients or animals treated with *x*-rays, helped to solve the problem. We can, however, say that it has been demonstrated that the blood of leukemic patients and of animals treated by *x*-rays contains a body or bodies capable of dissolving the leukocytes.

Conclusions. 1. At present it is best to consider myelogenous leukemia a malignant neoplastic disease.

2. The *x*-rays are effective in treatment as a palliative measure.

3. The action of the *x*-rays is associated with the production in the patient of a leukolytic body or bodies.

[15] Deut. Archiv f. klin. Med., lxxxiii, 1905.
[16] Berliner klin. Woch., xliii, Nr. 23.
[17] Jour. Exp. Med., January, 1907.

PYELONEPHRITIS COMPLICATING THE PUERPERIUM.

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When the upper urinary tract becomes obstructed and the contents of the accidental cyst thus formed become infected, four symptoms will be met with together or in succession—pain (localized in the lumbar region on the side of the lesion, or on both sides if the lesions are bilateral), vesical disturbances, a rise in temperature, and pyuria. Before considering this symptomatology in detail, I wish rapidly to sketch the usual manner in which the various pyelonephritides make themselves manifest after labor.

The commencement of the renal process may often be insidious and early. A patient does not do well during the first two days after delivery, each evening showing a slight rise in temperature, so that the physician immediately begins to suspect an infection of the uterus. Pain very infrequently indicates the renal lesion, and may not develop at the beginning of the process, so that the patient does not complain of the renal region. In other cases, the commencement of the disease may be very sudden and occur late in the puerperium. There will be a severe chill and a sudden rise in temperature, accompanied by marked pain. From recorded cases I think one may assume that the descending pyelonephritides are of rather slow development, while the ascending forms appear very shortly after infection of the lower urinary tract, as in the following case:

The patient, aged twenty-four years, had always been genitally well and had never suffered from any symptoms which could be related to the bladder or urethra. Married thirteen months ago, she had become pregnant. During labor, which was rather slow, the nurse was obliged to pass the catheter three times on account of vesical inertia. The family physician delivered the patient with low forceps after she had been twenty-six hours in labor. The child weighed 8½ pounds. After delivery it was necessary to resort to the catheter every six hours for the next twenty-four hours on account of the persisting vesical inertia. On the fourth day the lochia possessed a strong odor and was lessened in amount; the evening temperature showed a rise of 1.5°, so that intra-uterine irrigations were resorted to morning and evening. The evening temperature continued to increase, while in the morning the temperature never reached normal, and, as the lochia continued rather fœtid, the physician was inclined to diagnosticate a mild form of uterine sepsis, and I was asked to see the patient in consultation.

I saw her on the sixth day of the puerperium, and made the following notes: She had been passing her urine spontaneously, for the past three days. It was found cloudy, and the cloudiness appeared universally distributed throughout the test-tube. Upon palpation of the abdomen, deep-seated pain could be elicited in the right lumbar region, but the pain was not spontaneous unless the patient moved quickly in bed. The left lumbar region appeared in every way normal. On account of these functional symptoms, I was inclined to eliminate the diagnosis of uterine infection, and made one of right-sided pyelonephritis. There was no evidence of cystitis, although the bladder presented some tenderness on suprapubic palpation, but there was no pain on micturition.

On the eighth day the urine was passed in much larger amount, but was still turbid, and when allowed to remain in the test-tube the sediment composed about one-fourth of its bulk. This sediment was found to contain nothing but pus and large numbers of bacteria, which, by bacteriological examination, proved to be colon bacilli.

The treatment consisted in a milk diet, large quantities of water, and the administration of 0.5 gram (7.5 grains) each of salol and urotropin, three times daily. Four weeks after my first visit the urine upon microscopic examination was found perfectly normal and the patient had returned to her former health.

No matter how the renal affection may make its appearance, the following are the characters of each of its symptoms: The pain is seated in the lumbar region, topographically limited above by the border of the ribs, inwardly by a line drawn through the umbilicus, while it often happens that it radiates downward, following the direction of the ureter, as in nephritic colic. Laterally there is no fixed limit; the painful area consequently corresponds to what is ordinarily termed the flank. The pain is persistent in most cases, deep-seated, and paroxystic, and varies much in intensity. It is increased by movements on the part of the patient, and especially when bimanual palpation is practised, just as in the case of peritonitis or appendicitis. It does not seem to be increased by micturition, but any effort producing contraction of the abdominal muscles makes it appear. As to the time of its appearance during the evolution of the affection, nothing absolute can be said; but it usually begins with the renal process and ends when the latter is recovered from, while its diminution is notably evident when the ureter on the diseased side is allowing the passage of the pus, while, at the same time, and from this very reason, the temperature shows a tendency to subside.

The bladder disturbances may be considered under two heads. First, one may be dealing with a cystitis present before the development of the renal affection, or concomitant with it; under these circumstances there are all the classical symptoms of inflammation of the bladder, from which follows an ascending involvement of the

ureter and renal pelvis. In the second class of cases one is dealing with vesical disturbances without infection of the bladder, and this is what is more ordinarily observed. Therefore there are no real pains on micturition, excepting when a ureteritis has apparently been the factor in the production of the cystitis; there is no tenesmus and only a slight painful sensation when the organ is pressed upon suprapubically, which, for that matter, may be the result of extension of the pain along the ureter. This slight amount of participation, on the part of the bladder, in the pain resulting from pyelonephritis may be placed under the head of reflex cystalgia, which is symptomatic of some lesion involving some part of the genito-urinary tract. It should be recalled that the bladder is not easily inoculated by a dirty catheter, as I shall endeavor to point out later.

The third symptom is fever. This usually occurs as an evening rise, the morning temperature never falling to the normal. During the entire duration of the renal affection a rise in temperature need not of necessity be constant, because oftentimes it occurs in paroxysms dissociating the painful phenomena to which it may succeed, and, sometimes indicating their temporary or final disappearance. The chart will show from 39° to 40° C., or even more, because infection of the renal pelvis, under the influence of tension, will give rise to more fever than any other infectious lesion of the urinary apparatus. Although the temperature may remain high, the general condition of the patient is usually relatively good, and, although the tongue is coated, it remains moist and rose-colored, and not that bright-red tongue accompanied with marked dryness that is present in severe forms of infection. The pulse is rapid, and, although sometimes of high tension, it never becomes filiform. The mildness of these symptoms may be attributed to the fact that the intact kidney suffices for the elimination of the toxins. If delirium supervenes, it is rarely serious and is of very short duration.

Polyuria and pyuria are present. The urine is cloudy and in amount averages from two to two and one-half quarts in twenty-four hours. It is a pale yellow in color, cloudy, and more or less milky, while occasionally it may contain sand. The quantity of urine, which at first may appear diminished on account of the poor functioning of one of the kidneys, is in reality normal and not infrequently increased. This fact is explained by the law of compensation of symmetrical organs, and the well-known experiments of Strauss and Germont have proved beyond a doubt that the healthy kidney increases its functions and secretes a greater quantity of urine. Usually a small amount of albumin is present, this varying from 1 to 2 grams, while microscopically granular leukocytes in fairly large numbers accompanied by numerous epithelial cells, hyaline casts, and pus cells are to be seen. Bacteria are ordinarily present.

In pregnancy there are two undoubted pathogenic factors: (1) The uterus, according to the position that it assumes during the

second stage of its evolution, and as this tends more frequently to the right, it compresses the right ureter, while, should the organ lie toward the left, the left ureter is compressed, a condition of affairs relatively rare. The ureter becomes compressed by the uterus at the point where it passes over the bony projection of the superior strait at the level of the sacro-iliac symphysis. This pressure occurs more particularly during the last two months of pregnancy, and although certain German writers have considered that the child's head when engaging in the small pelvis compresses the ureters, I do not think that this is ordinarily the case.

When one ureter has become compressed, stagnation and retention in the renal pelvis result, placing the kidney in danger of infection, the chances of which are all the greater, on account of the special condition of auto-intoxication occurring in the pregnant female. It would be quite useless to waste time to demonstrate that a pregnancy, no matter how fortunate, lessens those natural resources necessary for combating the agents of infection. This agent, in the cases I am now considering, is usually the colon bacillus, either alone or associated with other organisms, whose preponderance over the former may sometimes be considerable.

We may consider whether in the puerperium, the three causes of pyelonephritis, the microbe, the soil, and the etiological cause, are active. As to the microbe, the researches of Reblaud, Weiss, and Weill show that the colon bacillus is particularly prevalent in puerperal pyelonephritides, and this has been found to obtain in my own cases. The organism may reach the kidney either by descending infection or by ascending infection. In those cases in which no digital examination or instrumental interference has been resorted to on the lower urinary tracts, in which there is neither a former or coexisting cystitis or urethritis, it is only possible to admit that the starting point of the renal infection is the intestinal mucosa.

To make this assertion more evident, I would record the following case: The patient, a primipara, aged twenty-seven years, was seen in consultation, the following notes being furnished by her family physician: The labor lasted fourteen hours, and was in every way normal. The child was being breast fed. Twelve days after labor I was asked to see the patient. For several days she had been complaining of pain in the right lumbar region, accompanied by marked constipation. The temperature was 39.2° C.; pulse, 115. Her face had an anxious look, one of infection, and the tongue was heavily coated. Palpation from the lumbar region on the right side almost to the iliac fossa gave rise to considerable pain, and, on account of the muscular rigidity, the examination was rendered difficult. There were no bladder symptoms. The lochial discharge was normal. Examination of the urine voided at my visit showed a cloudy, straw-colored fluid, which after being centrifuged

showed an abundance of leukocytes, bacilli (colon bacillus?), and a few epithelial cells, probably from the renal pelvis.

Bimanual palpation of the uterus and adnexa revealed nothing abnormal, so that all suspicion of uterine infection could at once be done away with and a diagnosis of right-sided pyelonephritis was made. The treatment consisted of an absolute milk diet and 1 gram (15 grains) of urotropin four times daily.

The patient was seen three days later, when it was found that the evening temperature and the pulse were the same as upon my first visit; and the patient complained of a frequent desire to urinate. Bimanual vaginal examination increased this desire, and by a more careful palpation it at once became evident that the right ureter was painful along its entire length. There was less pain in the lumbar region, so that palpation of the kidney was rendered easier, the lower pole of the organ being distinctly felt, and when caught between the fingers gave rise to rather sharp pain. The same treatment was continued for five weeks, during which time the pulse and the temperature gradually returned to the normal, and the pus diminished in quantity, so that at the end of this time there was barely a trace, and the bacteria had entirely disappeared. Some months afterward I was informed by the physician that the patient had entirely regained her health.

Before affirming the reality of a descending infection, one should not forget that during labor, and especially at the time of delivery, entrance of bacteria into the urethra, whose mucosa becomes everted, is rendered easy by the eversion of the anal orifice.

A former cystitis, acute or chronic, latent or awakened, related or not to pregnancy, a primary or secondary infection produced by a dirty catheter, commencing in the bladder or propagated to the ureter, represent the various ways in which Bacterium coli may reach the kidney. When ascending infection is incriminated, it is not even necessary for the bladder to become infected before a ureteritis becomes established. The organism may remain in the bladder, leading a saprophytic life, and then extend upward along the ureter, this being favored by a temporary retention; then it enters the dilated ureter and renal pelvis, here encountering a point of lessened resistance, which is favorable to its development, the contrary being the case of the bladder.

The ascending infection has been doubted by many observers, who believe that such infection is probable only when there is absolute evidence of a vesical infection. But, on the other hand, what proves the reality of an ascending infection is that, in some instances, the same organism, primarily located in the kidney, is continually thrown into the bladder with the urine, and still no infection of this viscus takes place, while in other cases the organism enters the bladder, causing an ascending infection of the ureter and kidney without producing any infection of the urinary reservoir. Then,

again, it is well known that, in certain infections, the bladder constantly contains Bacterium coli, from the fact of retention, and still cystitis rarely develops. All this goes to show that Bacterium coli can pass through the bladder without leaving behind any traces of its presence that are clinically demonstrable.

Bacterium coli is far from being the specific agent of postpartum pyelonephritides, because it is the common factor of renal disturbances during pregnancy as well; this brings me to the question of the soil in which the pathogenic germ may grow. Clinically speaking, a woman who has just passed through labor represents a surgical case; there is a wound of the cervix, more or less extensive, and at least more or less contusion of the vagina and perineum. Without considering the loss of blood due to the raw surface left by the placenta, it should be recalled that the lochia represent an excellent culture medium for various bacteria. Intestinal atony, which is of frequent occurrence during pregnancy, reaches its highest point of intensity after delivery, no matter what may be its cause. This very constipation retains the intestinal bacteria within the gut, so that the infectious agents may easily enter the blood current, and in the cases I am now considering it has been shown that Bacterium coli can enter the kidney by the renal artery. Vesical inertia does not have as bad an influence on the organism as intestinal atony, but it is a very marked predisposing cause for infection from the catheter or other bladder instrumentation.

If the patient nurses the child, it is evident that this weakens the organism generally on account of the removal of a certain amount of nutritive substances which go to manufacture the milk. In other words, the new mother may be looked upon as not in absolutely normal health for the above-mentioned reasons, and from this very fact, she is more susceptible to develop morbid phenomena if the occasion presents itself.

The cause of pyelonephritides occurring in the puerperium may be summed up briefly as follows: Labor is over and the uterus is about to commence its arduous work of retrogression, the maximum activity of which is probably between the ninth and twelfth days. Measurements taken by Varnier, Zinsstag, and others have shown that, the bladder being empty, the height of the uterus above the pubis is 13 cm. the second day postpartum, 6.5 cm. on the ninth day, and 5.5 cm. on the twelfth day. This decrease in height is due to a diminution of the uterus resulting from an incomplete atrophy of all the elements forming the uterine walls before pregnancy which have become hypertrophied by gestation; secondly, there is anteflexion of the uterus. The apparent regression is greater than in reality, and is principally due to an exaggerated uterine anteflexion, because after the fifth day the uterus will have taken on the shape of a non-pregnant organ, its corpus, being directed according to the axis of the superior strait, is perpendicular to the vagina.

Now, at this time, as the corpus uteri is nearly in the axis of the superior strait, it can compress those organs which lie behind it, and this is all the easier from the fact that after labor most women are kept in a horizontal position, a condition which pushes all the abdominal viscera backward. Now, the ureter passes exactly over the sacro-iliac symphysis, and a very mild compression can result in its complete obliteration. According to the experiments of Ludwig and Löbel, the pressure of the urine in the renal pelvis and ureters is always very low, and does not appear to reach above 10 mm. of mercury. This conclusion has been confirmed clinically, two cases of hydronephrosis due to compression of the ureter by an abnormal branch of the renal artery having been recorded by Le Dentu, while Young, of Baltimore, not long ago, published the case of a stricture of the ureter resulting from a mere compression by an inflamed seminal vesicle. Now, the weight of a uterus undergoing involution varies between 300 and 600 grams from the eighth to the fifteenth day postpartum. By its convex posterior aspect it lies on the ureters, which are dilated from the recent pregnancy, as has been proved by numerous writers, and having incompletely returned to their normal caliber, a fact which increases their surface of compression, no matter how small may have been this temporary dilatation. For that matter, this compression of the ureters does not belong to pregnancy and the puerperium alone, because it may result from any kind of pelvic tumor, and we all know that there are many renal complications arising during the progress of uterine carcinoma resulting from compression or obliteration of the ureters from the neoplasm. Uterine fibromas, ovarian cystomas more rarely, may compress the ureters and be the starting point of renal lesions. Consequently, during the puerperium, the uterus may compress the ureters and thus set up a pyelonephritis.

There are also two other ways of explaining the development of this renal complication, the first being the persistency of a condition of lessened resistance created during pregnancy by compression of the ureters by the pregnant uterus; in this respect, I believe the following case may be taken as an illustration: The patient, married at twenty-six years of age, became pregnant twenty-one months later. The pregnancy, during the latter months, was complicated by phenomena of auto-intoxication, although the urine remained perfectly normal. The patient complained of rather severe headache, marked constipation, œdema of the ankles, and disturbances of sight. For this reason, she was at once put upon an absolute milk diet, which resulted in a notable diminution of the symptoms. The uterus was unusually developed and lay decidedly to the right side of the abdomen. Labor occurred at term, and was perfectly normal.

The first few days following labor presented no incident worthy of note; micturition was normal. On the sixth day the temperature,

which had been perfectly normal, suddenly rose in the afternoon to 39.5° C., accompanied by a severe chill. The tongue became coated, the abdomen somewhat distended and generally painful, but a careful examination showed that the greatest amount of pain could be elicited in the right renal region. An examination of the urine at this time showed that it contained some pus cells and quite a number of red blood corpuscles; also some bacteria. As far as the uterus and adnexa were concerned, they were absolutely normal, the lochia having its accustomed odor and being discharged in proper amount. Two days later the urine became decidedly purulent, and the pain became localized in the region of the right kidney; consequently a diagnosis of pyelonephritis was made. During the next week the amount of pus in the urine increased; red blood corpuscles were also present, and bacteria, which in the hanging drop appeared like the colon bacillus, were evident. The temperature at the end of the week had reached 40.2° C., and the pulse was becoming filiform and reached 120. The patient presented all evidences of having a large focus of pus in the abdomen, and it was, therefore, decided to cut down on the right kidney. This was done the following morning by transverse lumbar incision, and a large congested kidney with a considerably dilated pelvis was exposed. The organ was split open, and when the interior of the pelvis was reached a large phosphatic calculus was removed, which, after drying, weighed 47 grams, while the renal pelvis contained 50 c.c. of distinctly purulent urine. Other than the congestion the renal parenchyma appeared to be normal. Lumbar drainage was established and at the end of three months the patient was discharged well.

From most of the published cases it will be seen that pyelonephritis of pregnancy and the puerperium is usually right-sided, this being easily accounted for by the fact that the gravid uterus is more prone to lie toward the right, while it rarely becomes inclined toward the left. Cases of left-sided pyelonephritis, signifying that the ureter must have been involved in the first place, may be explained by the fact that the left ureter has been compressed during pregnancy, or that there is a continuation of the compression by the uterus undergoing subinvolution.

Although, during pregnancy, a differential diagnosis is most urgent, on account of the unfortunate influence that pyelonephritides may exercise over the mother and especially the child, it is, perhaps, quite as imperious during the puerperium, and here, certainly, it is much more difficult, because uterine infection is more likely to come to one's mind, in addition to appendicitis, typhoid fever, or some intestinal infection. In these cases, in which all the above-enumerated symptoms are present, especially pus in the urine, it seems that the solution of the problem should be easy, although uterine infection may be also present with ureteral and renal lesions without there being any connection between the two morbid processes.

Some diseases may simulate pyelonephritis, such, for example, as acute tuberculosis or typhoid fever, but I shall only here consider puerperal infection, appendicitis, and intestinal infection.

In considering the most important of these, puerperal infection, abdominal pain, elevation of the temperature, and a bad general condition are the symptoms common to both pyelonephritis and uterine infection. When these symptoms are met with, the first diagnosis that comes to one's mind naturally is puerperal infection. However, there are several characteristics of these symptoms which weigh in favor of pyelonephritis. These are the marked rise in temperature of 2° or 3° C., followed by a morning drop to the normal, or even below. The condition of the pulse is never in relation to the rise in temperature, while in puerperal infection the pulse follows the temperature curve, and is usually poor. In pyelonephritis the patient's general condition is good during the intermission of the temperature. There is nothing absolute, of course, in these differences, but they help to differentiate the diseases in many instances. When a kidney contains a certain amount of pus pent up in the pelvis, after a while the temperature and pulse will be as in any serious form of infection, and this was the case in the instance of pyelonephritis with calculus reported.

Pain in the lumbar region is not very marked in ordinary cases, and, therefore, the patient is not likely to complain of it. But all things taken into consideration, a differential diagnosis may be made even before the appearance of pus in the urine, if one will only recall pyelonephritis; this, as many other affections is not diagnosticated simply because it is completely forgotten. As a practical conclusion of what has been said above, given elevation of the temperature during the puerperium, one should commence by a careful examination of the uterus and adnexa, and, having found that there is no lesion in the small pelvis, the possibility of a pyelonephritis should at once come to one's mind; a careful examination of the urine should be made, as well as a methodical palpation of the renal region and ureters, although the patient may be perfectly free from pain in these regions.

The next important differential diagnosis tc make is that of appendicitis and pyelonephritis. If in a puerperal woman examination reveals nothing abnormal in the uterus and adnexa, and abdominal pain is present, with a rise in temperature and constipation (a frequent occurrence during the postpartum), the appendix should be immediately examined; not infrequently the region of McBurney's point will give rise to pain on palpation, from the fact of reflex pain along the ureter. The maximum point of pain will be found close to McBurney's point, for the simple reason that the course of the ureter is very near the former, and consequently an erroneous diagnosis of appendicitis may be made. I have not in mind here the very acute forms of appendicitis which are ushered in

by vomiting, high fever, peritoneal expression, and intense pain in the cecal region, with almost complete intestinal occlusion, because, under these circumstances, the diagnosis of the appendiceal lesion would be only too evident. In those cases in which there is any doubt as to the diagnosis of appendicitis, one should wait, always prepared to interfere should any change in the symptoms require operation. One will not be obliged to wait long, because a careful examination of the urine, kidneys, and ureters will quickly do away with the diagnosis of appendicitis, while that of pyelonephritis will be made. In passing, I would simply refer to those ordinary forms of intestinal infection which arise during the puerperium in women having chronic constipation. In these cases, as in pyelonephritis, the tongue is coated, there is a rise in temperature, and the patient complains of abdominal pain, but, here, again, careful examination of the urinary apparatus will soon settle the question of diagnosis.

A few words as to the examination of these cases. Palpation, which during the puerperium is much easier and gives better results than during pregnancy, will only give rise to one symptom, namely, pain. The renal pelvis and its ureter must reach quite a high degree of dilatation in order to be felt by bimanual palpation. One rarely, if ever, meets with these instances of calculous hydronephritis of long duration, and one is often struck by the contrast existing between the violence of the general reaction and the absence of any important tumefaction in the lumbar region. Ordinarily there is some muscular rigidity, but only on the diseased side; this is especially manifest when the intrarenal tension has become considerable, and it ceases or diminishes when the renal pelvis becomes emptied, thus lowering the tension. Vaginal examination per se gives little information, because the ureter is usually healthy in its intravesical portion, although in one of my cases it was distinctly palpable. But even when the ureter is not involved in its terminal portion, pressure made upon it near its entrance to the bladder may provoke a ureterorenal reflex, and occasionally, when all other signs are wanting, this alone has allowed one to make a diagnosis of pyelonephritis. The urine will always be found to contain pus, and the pyuria alone should be sufficient to indicate a pyelonephritis if the bladder itself is not the seat of any lesion. A symptomatic cystalgia may be differentiated from a true cystitis by the presence of a cloudy polyuria, absence of bladder pain on palpation, and by a normal sensitiveness to tension, because one should be able to inject from 150 to 200 c.c. of liquid into a normal bladder without setting up a desire to urinate. I would here point out that the amount of suppuration is in no way proportionate to the intensity of the infection; not infrequently the urine may be almost limpid, although the general symptoms are quite serious. Generally speaking, when the urine becomes very purulent the

temperature falls and the pain diminishes, from the simple fact that drainage is effective.

Ureteral catheterism and intravesical segregation of the urine are very important in order to ascertain if one or both kidneys are involved, and, during the puerperium, their undertaking is far less difficult than during pregnancy.

If, as I have endeavored to show, pyelonephritis occurring during the puerprium should be particularly differentially diagnosticated from puerperal infection, it may be asked whether or not the former, on account of the extension of the pathogenic bacteria giving rise to it, may not produce the latter. Consequently, the prognosis of pyelonephritis manifesting itself during the puerperium should be considered from the point as to whether or not the urinary infection may finally involve the genital apparatus, secondly, from the renal viewpoint, and, thirdly, from the question of nursing.

Referring to the first question, it will appear from cases recorded thus far that patients having pyelonephritis after labor are in no way contaminated, as far as their genital organs are concerned. This fact might, in the first place, appear strange when one considers how easily inoculation of the vagina and uterus might take place from the pyogenic agents contained in the urine. Perhaps the non-infection may be due to certain chemical differences between the purulent urine and the postpartum uterine and vaginal secretions, preventing the development of the bacteria contained in the urine. Then, again, it may be due to a careful protection of the genital zone in patients presenting urinary infection. However this may be, it seems to me, clinically demonstrated that the uterine and vaginal lesions of continuity arising during labor do not become infected by the urine. From the view point of acute renal infection, a cure is generally the outcome, but relapses of the renal process are prone to arise.

Two therapeutic measures can be resorted to in dealing with a pyelonephritis, whether during pregnancy or postpartum, namely, a medical and a surgical treatment. No comparison can be made between the two, because they have their own special indications, surgery being resorted to only in complicated cases.

The medical treatment consists essentially in rest in bed, a milk diet, and alkaline drinks combined with diuretics and urinary antiseptics. To relieve pressure on the ureters from the uterus, various positions to be assumed by the patient have been advised. Excellent results have been obtained by having the patient lie on the side opposite to that of the involved kidney, thus causing the uterus to fall over to the other side and away from the ureter which it compresses. Whether the lesions be on one side or bilateral, Lepage advises the patient to sit in a chair rather than lie in bed, because in the former position the uterus no longer brings all its weight to bear on the ureters and, consequently the compression is diminished.

For the infection of the kidney and ureter an absolute milk diet must be instituted. Salol, urotropin, and the balsams have their indications. When there is retention with infection, some favorable results may be obtained by distention of the bladder, as advised by Pasteau. It is well known that distention of the bladder produces an increase of the renal secretions, as well as of the contractility of the excretory apparatus. Therefore, Pasteau injects from 150 to 160 c.c. of tepid boric acid solution very slowly into the bladder; this is repeated three times a day, the patient being requested to retain the fluid in the bladder as long as possible. It should be pointed out that this treatment is absolutely contra-indicated when cystitis is present.

As to surgical treatment, nephrotomy is probably the only operation required, and from an examination of the literature it appears that it has rarely been necessary to resort to it. Nephrectomy, which is an extreme and radical resource, has never been required in cases of pyelonephritis occurring postpartum, so far as I am aware.

THE ANATOMY AND PATHOLOGY OF THE CAROTID GLAND.

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This work was undertaken primarily to determine the effect of various systemic diseases on the carotid gland; incidentally, the anatomy and histology of the gland have also been closely observed. A review of the literature was given by Kohn,[1] in 1900, and by Funke,[2] in 1904. A summary of the cases of tumors of this organ, reported up to the time of their publication, was given by Keen and Funke,[3] in 1906. Therefore, it does not seem at present necessary to go over the literature, and I shall proceed to a description of my own findings.

Material and Technique. Human material was obtained as fresh as possible from the cadavers at necropsies performed in various hospitals of Chicago, and particularly the Cook County Hospital. Fifty human cases altogether were examined; and from animals killed at the Hull Laboratories and at Armour & Co.'s Union Stock Yards, material was taken for comparative study. Fixation was by Zenker's fluid.[4] In the first 22 human cases, and

[1] Archiv f. mikro. Anat., 1900, vol. lvi, p. 81.

[2] American Medicine, 1904, vol. viii, p. 109.

[3] Jour. Amer. Med. Assoc., 1906, vol. xlvii, pp. 469 and 566.

[4] Kohn recommends a mixture of formalin and potassium bichromate. Staining was done with Delafield's hematoxylin and eosin, Van Gieson's, and Mallory's reticulum stains.

in all animal material, pieces of tissue about an inch in average length, including the common, internal, and external carotid arteries, with some branches of these, were embedded in celloidin. The tissues were then cut at various levels of about 100 microns intervals, cutting three or four sections at each level, of about 12 microns in average thickness. The sections were stained with hematoxylin and eosin and search was made for the gland. On cutting blocks of tissue in this way, in many cases it was possible to tell by the naked eye whether the gland was at the level reached; it showed itself as a clump of tissue closely related to the outer coat of the arteries, pinkish white, sometimes lobulated, sometimes uniform, in appearance. Three kinds of tissues were constant sources of confusion: (*a*) A ganglion of the sympathetic nervous system, which is usually uniformly whitish in appearance, but is not closely adherent to the adventitia of the carotid bloodvessels. (*b*) A nerve trunk, the fasciculi of which on cross section resemble the lobules of the carotid gland; this nerve trunk was either the vagus or sympathetic; in the first instance it was not attached closely to the outer coat, and in no case was it found posterior or anterior to the bifurcation of the carotid artery, but occupied always the lateral side. When the nerve trunk was sympathetic it was sometimes cut obliquely, so that a lobular formation was not shown; it resembled a clump of connective tissue, which was not adherent, however, to the adventitia. (*c*) A clump of fibrous connective tissue, which is adherent to the adventitia, but differs generally from the carotid gland by the dead white color and its uniformly dense appearance; in several cases, however, the gland presented exactly the appearance described, and only microscopic examination decided the real nature of the tissue.

Therefore, in macroscopic examination of blocks of tissue cut transversely the points of differentiation were (1) pinkish white color; (2) lobulation or uniformity; (3) close relationship to adventitia, into which the capsule of the carotid gland seems to merge. The presence or absence of the gland was considered established only after a careful microscopic examination of the stained sections.

This method of finding the gland is very valuable, since sometimes the gland, either because it was very small or was concealed in the adventitia, escaped detection by macroscopic dissection. It is the only reliable method for finding the gland in lower mammals, since in these it is relatively small, and the surrounding connective tissue is much thicker and denser than in man.

Beginning with Case XXIII, the attempt was made to dissect out the gland, and in almost every case this could be successfully done. When it was located, either the gland alone or a piece of bloodvessel with it was embedded in colloidin or paraffin. In dissecting out the gland fresh material was always used, because hardening fluids tend also to empty the bloodvessels and capillaries and coagulate the tissues; thus the reddish appearance could not be well dis-

tinguished. It is not uniformly red, but rather mottled with light red and dark red points. In this dissection the gland is again to be distinguished from (*a*) connective tissue, which is paler, uniform, and can be broken into fibers; (*b*) lymph glands in the intercarotidal tissue, which are sometimes hyperplastic, although usually they cannot be seen. If they are hyperplastic, they are usually uniformly red in appearance and detached from the wall of the bloodvessels. In one case the carotid gland was found hanging from the external carotid artery by a fibrous stump, and was so surrounded with fat that it appeared very much like the lymphatic glands (which were enlarged in this case probably on account of tuberculosis of the lung); only its attachment to the artery guided me to keep the tissue for further examination, and to my surprise microscopically it turned out to be the carotid gland. (*c*) It also has to be distinguished from ganglia of the sympathetic, which are, however, of the white color of the nerve, are connected with the sympathetic cord, and not attached to the adventitia of the carotids.

ANATOMY AND TOPOGRAPHY. The carotid gland is generally described as a reddish ovoid organ of firm consistency, the longest axis being vertical, the upper pole broader than the lower, lying on the posterior surface of the bifurcation of the common carotid artery, and not constantly present in man. Funke states that he found it most frequently on the inner and posterior side of the internal carotid. Schaper[5] states that the gland is spindle-shaped, and that in man it is more regular in size and shape than in other mammals. Its size varies, according to different authors: Luschka[6] gives it as 7 by 4 by 2 mm., Schaper as 5 to 7 by 2, 5 to 4 by 1.5 mm. A fibroelastic stump connecting the gland with the bloodvessel on which it lies, and carrying its artery, is described as the "ligament of Mayer." Most authors agree that there is only one nutrient artery springing from the one of the carotid arteries on which the gland lies. Henle[7] states that it receives two or three branchlets from the *carotis primitiva* (common carotid). Poirier and Charpy, speaking about the relation of the gland to the nerves, state that a great number of filaments surround it, either directly or after forming a plexus, the intercarotidal plexus, the filaments of which come from the superior cervical sympathetic ganglion, the *nervi molles* of Haller, the glossopharyngeal, hypoglossal, and superior laryngeal.

In my own experience I found the shape of the gland generally ovoid, in several cases rounded, and in a few elongated. In the majority of cases it was found single on each side of the neck. Luschka has observed the occasional splitting of the organ into two lobes, and my findings confirm his statement. The gland on the left side in Case XLI was remarkably flattened, and on the free edge there was

[5] Archiv f. mikro. Anat., 1892, xl, p. 287.
[6] Archiv f. Anat. u. Physiol., 1862, p. 406.
[7] Cited by Poirier et Charpy, Traité d'anatomie humain, Paris, 1901, tome ii, p. 668.

a depression, which, however, was not deep enough to make the gland a uniform shape. In Case XXIX, on the left side, the gland was elongated and constricted at the middle, dividing it into two lobes

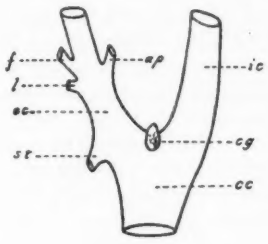

FIG. 1.

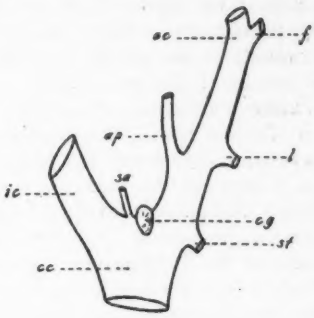

FIG. 2.

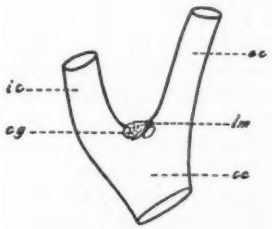

FIG. 3.

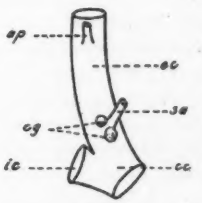

FIG. 4.

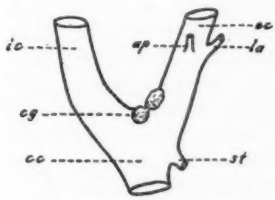

FIG. 5.

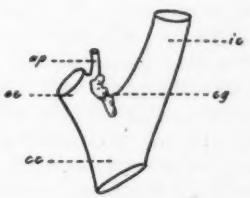

FIG. 6.

The carotid gland, posterior views, illustrating its gross anatomy and variations in location. Fig. 1. The most common form. Figs. 2–6. Several other types described in the text. *Cc*, common carotid artery; *Ec*, external carotid; *Ic*, internal carotid; *Sa*, small anonymous artery; *Ap*, ascending pharyngeal; *St*, superior thyroid; *L*, lingual; *F*, facial; *Cg*, carotid gland; *Lm*, ligament of Mayer.

united by an isthmus (Fig. 5). In Cases XXXV (Fig. 4) and XXXIII there were two distinct and well defined bodies on the left side. The gland was measured macroscopically every time that it was possible to do so, by carefully dissecting out the fat, including the connective tissue capsule and the glandular tissue in the mensuration. My measurements agree with those of other authors as to the variability of the gland in size. Out of twenty-six measurements, the average is 5 mm. in length, 3 mm. in breadth, and 2.5 mm. in thickness—an average diameter of 3.5 mm. The largest gland was 8 by 5 by 4 mm. The smallest was not detectable by naked eye inspection; only under the microscope was its presence established, and it was 0.5 mm. in diameter.[8]

As to the position of the gland, I also found it most commonly between the internal and external carotid arteries, resting posteriorly on the bifurcation of the common carotid, the ligament of Mayer being attached to the artery on which it lies (Fig. 1). Variations from the usual location are the following: In Case XXIV, on the right side, the gland was hanging by the ligament of Mayer to the outer side of the external carotid, 3 mm. above the bifucration; in Case XXXI, on the left side, it was placed in front of the ascending pharyngeal artery, which arose at a lower level; in Case XXVIII, on the right side, it was a little elongated, placed upon the middle, not behind the bifurcation; in Case XXXVII, the right side was elongated, resting on the groove of the bifurcation superiorly and posteriorly, its upper pole being in close relationship with the ascending pharyngeal artery and deviating somewhat to the right of this, the lower pole resting posteriorly on the bifurcation (Fig. 6). In several other cases it was more intimately attached to the external than to the internal carotid. In one case the ligament of Mayer was not attached to the artery on which the gland was lying; the gland resting on the inner surface of the internal carotid about 1.5 mm. above the bifurcation, while the ligament of Mayer, with its contained bloodvessel, stretched across the intercarotidal space, to be attached to the external carotid, into which the carotid gland artery opened (Fig. 3). In 5 cases (on only one side in 3 cases, and on both in 2) a small artery sprang from the bifurcation of the common carotid, and in every case the gland was intimately related to it, either embracing or directly adjacent to it (Fig. 2). Of the two lobules of Case XXXV, the one was anterior, the other lateral to this small artery (Fig. 4). I am not aware of any name given to this artery.

The gland was found only once in 8 human fœtuses examined by Funke, who apparently examined no adult material. Out of 50 human cases of my own it was found present in all but 5. In 4 cases it was found only on one side, and in 1 of these last cases the only

[8] The gland increases in size as the individual grows, from proliferation of the parenchyma, bloodvessels, and connective tissue.

carotid body present was much enlarged (about 5 mm. in average diameter), as though a compensatory hypertrophy had taken place.

Speaking of the comparative anatomy of the carotid gland, Schaper[9] concludes: "It is probably present in all mammals. A homologous organ is not found in birds, at least in the neighborhood of the bifurcation of the carotid artery. The so-called carotid gland of batrachians and salamanders is not homologous with the one in mammals. In fishes it has not yet been observed, but the *Axillarherzen* that Rochen and Haien found, and about which von Leydig wrote, are probably of the same nature." Kohn's findings confirm Schaper's conclusions. Funke was not successful in finding the gland in lower animals (cat, rabbit, and guinea-pig).

By taking pieces of tissue containing the bifurcation of the carotid artery and cutting sections at 100 μ intervals, I was able to find it in cats, dogs, oxen, goats, and monkeys. In these animals the gland in every case was too small for macroscopic dissection, and the connective tissue capsule was so thick and dense that no characteristic external appearance of the gland is shown. I agree with Schaper as to the variability of its position in mammals; in general it is placed posterior to the bifurcation, more closely attached to one artery than to the others. In the ox the gland was found somewhere in the neighborhood of the division of the common carotid artery into internal and external maxillary and occipital arteries,[10] and it was divided into two separate lobes.

Embryology and Histology. There are three theories as to the embryology of the carotid gland (see Keen and Funke): (1) Endodermal, which holds that the gland is derived from the third or fourth branchial cleft, as is claimed by Stieda, Rabl, Pierre du Meuron, and others. (2) Endothelial, as is believed by Katschenko, and confirmed by Paltauf and Szymonowickz, from the endothelium of the carotid artery, the first sign of the gland being a cellular thickening in the adventitia of one of the bloodvessels. (3) Nervous, propounded by Kohn, on account of his having found chromaffin cells corresponding to those in the sympathetic ganglia, and he says it belongs to a category of its own. The nervous origin was long ago suspected by Luschka, on account of its close relationship to the sympathetic nervous system, thus resembling the pituitary body, the suprarenal gland, and the coccygeal body.

Luschka first described the histological structure of the carotid body, and concluded that it is glandular, made up of tubules and vesicles, the formed elements being molecular masses with naked nuclei and cells, the nucleus being partly dark, partly granular, with a nucleolus, the cells being mostly elongated, and also polygonal or irregular, showing sometimes great similarity to cylindrical epi-

[9] Archiv f. mikro. Anat., 1892, xl, p. 287.

[10] Martin. Lehrbuch der Anatomie der Hausthiere, 1904, vol. ii.

thelium. Arnold,[11] on the other hand, held that the gland is of glomerular structure, and states that the tubules and vesicles of Luschka were only apparent; he believed free cells, nuclei, and molecular masses were not present.

Both authors were partly right, however, as is found by recent investigations, and now it is considered established that the gland is made up essentially of two parts: (1) Capillary bloodvessels, richly anastomosed, forming a sort of tuft; and (2) cells, epithelioid in character, situated in the interstices limited by the bloodvesels. The gland is surrounded by a dense fibrous (white and elastic) connective tissue capsule, from which prolongations are sent in, dividing the gland into lobules, which are again divided by delicate strands of connective tissue, inclosing the "Zellballen" of Schaper. It is maintained by Marchand[12] that there exists a hilum structure where the artery enters the gland, and then gives up one branch for each lobule, which in turn is broken up into capillary glomeruli, around which the formed elements are arranged. The efferent vessels from these tufts are gathered at the periphery of the organ into wide, thin-walled veins. The capsule contains both medullated and non-medullated fibers. Ganglion cells are scattered and few in number. Paltauf[13] found a connective tissue reticulum, which is regarded as a postmortem alteration by Schaper.

The formed elements are described as cells with cuboidal, polygonal, sometimes irregular, outline, similar to epithelium, with rounded or oval intense staining nucleus, often eccentrically placed, and containing a nucleolus. Mönckeberg describes an angular form of nucleus. The protoplasm is somewhat granular and small in amount. Frey[14] likens the parenchyma to the plasma cells of Waldeyer. I have observed very carefully the cells stained with hematoxylin and eosin and fixed in Zenker's fluid, and two types of cells seem to be evident in most cases. The protoplasm of each kind is similar in character, and they differ principally in the character of the nucleus. In one (type I) this is large, rounded or ovoid, vesicular, with chromatin granules distinct, and a dot of chromatin or nucleolus at the center. In the other (type II) the nucleus is smaller, similar to the lymphoid cell nucleus, rounded, dense, uniformly dark blue stained, with the distinct chromatin granules. In the majority of cases the cells of the second type predominate, in a few those of the first. In cases in which the cells were only of one type, the second type was the one present.

Among the cells of the "cell balls" and in the stroma, Stilling[15] found a kind of cell which stains brown by neutral salts of chromic acid. Kohn confirmed Stilling's observations, and calls these

[11] Virchow's Archiv, 1865, vol. xxxiii, p. 194.
[12] Virchow's Festschrift, 1891, vol. i, p. 537.
[13] Ziegler's Beiträge, 1892, vol. xi, p. 266.
[14] Cited by Paltauf.
[15] Cited by Kohn.

"chromaffin cells." Similar chromaffin cells are present in the adrenal, and some authors consider the chromaffin substance to be the cause of the effect of the gland extract upon blood pressure.

Recently Mulon[16] succeeded in producing a rise of arterial blood pressure, and sometimes an acceleration of the beat and force of the heart, by injecting watery extracts of the carotid gland of horses into the viens of rabbits, and he attributes this effect to the chromaffin cells of the gland. I tried on a cat two injections of glycerin extracts of equal quantities of material from human carotid glands, and I found invariably a *lowering* of blood pressure. These experiments will be repeated as soon as a suitable amount of material is obtained, and a more detailed account will be published later.

Concerning the relationship between the parenchyma cells and the capillaries, authors divide themselves, Reclus and Chevassu[17] say, into two sides. Some, with Eberth, Marchand, and Paltauf, see between the cells and the capillaries a very intimate relationship, the epithelioid cells being outgrowths from the vascular epithelium, and cite the endothelial origin of the gland as a support of their contention. Others, with Luschka and Stilling, consider that between the vessels and cells there is a true independence; they liken the carotid body to the suprarenal gland, the carotid body deserving at all points the name of blood vascular organ, and cite the endodermal origin of the gland for their support. Kohn says it is neither a gland nor entirely an epithelial structure, but belongs to a category of its own.

In my own cases, when the capillaries were found greatly distended with blood, the relationship belween the epithelioid cells and the capillaries was more intimate, and they appeared to line the capillary lumina directly. When the bloodvessels were empty, however, fine fibrils of connective tissue seemed to separate the epithelioid cells from the endothelial lining of the capillaries.

In lower mammals Schaper found that the connective tissue of the carotid gland is less in amount than in the human, and he likens them to the younger stages of human carotid body. Kohn[18] describes four types of carotid gland, according to the degree of development of the connective tissue and the degree of division of the parenchyma. (1) "Compact type," a good example of which is the organ of the cat, which is characterized by fine interstitial connective tissue, so that the cellular appearance is more pronounced and a more parenchymatous constitution is shown. (2) "Lobular type," in which a greater amount of connective tissue divides the gland into lobules, pronounced in apes (*macacus rhesus*). (3) "Granular type," in which further subdivision of lobular into small granules (Zellballen) occurs, through an increased amount of connective

[16] Archives gén. de médecine, 1904, p. 3265.
[17] Rev. de chir., 1903, vol. xxviii, p. 157.
[18] Loc. cit.

tissue; this is the human type. (4) The "diffuse type," in which the cells are arranged in small globular groups, or small short strings, lying apparently independent of each other in the connect-tissue; this type is present in rabbits.

My findings agree with those of Schaper in regard to the relative amount of connective tissue. In addition, it was noticed that the carotid gland of the mammals observed contained only one kind of cells, cuboidal in shape, with the nucleus large, rounded, and vesicular, showing distinct chromatic granules and a nucleolus; hence, they correspond to type I of the cells found in human glands. In general it may be said that the carotid gland of the cat, dog, goat, ox, and monkey in my cases had more of a parenchymatous appearance than in man. In the goat, monkey, and cat it is of a more compact type. In oxen the gland appears of a granular type, little individual groups of cells in more of an alveolar arrangement, and separated by fine strands of connective tissue. The dog's carotid gland has a greater development of connective tissue, and approaches to the young human gland in appearance, while the others (goat, monkey, cat, and ox glands) are more parenchymatous than in young stages of the human gland. In monkeys, at about the bifurcation of the carotid artery, a lymphatic gland is constantly found, which was at first thought to be the carotid gland.

PATHOLOGY. Only tumors of the carotid gland have been reported so far. I am not aware of any description of other diseases of this organ.

Tumors. The increasing number of cases reported within the last few years, and the possibility of mistake in the diagnosis of tumors of the neck, make one suspect that these tumors are not so infrequent as was formerly believed. Keen and Funke collected 29 cases: 27 observed in the living, and 2 discovered on autopsy. Hutchinson's[19] 8 cases of "potato tumors" are probably newgrowths of this kind. Their etiology is unknown; some coincidences with other diseases, such as tonsillitis in one case, buccal phlegmonous inflammation following extraction of a tooth in another, have been mentioned in attempting to indicate the ultimate exciting causes, but there does not seem to be enough foundation to attribute any special significance to these facts. There is no sex influence. They occur particularly in adolescence and adult life, cases having been observed from the eighteenth to the sixtieth year, mostly about the thirtieth. V. Heinleth[20] attempts to explain their formation by stating that the carotid body develops until puberty, when it either atrophies or development is arrested; if it continues to grow, a tumor is formed.

The tumors are usually oval in shape, the size varying from a pigeon egg to a goose egg. The consistency is variable, but it is

[19] Illustrated Medical News, October 18, 1888, and also November 3, 1888.

[20] Münch. med. Woch., 1900, vol. xlvii, p. 899; and also Centl. f. allg. path. u. Path. Anat 1900, vol. xi, p. 599.

usually moderately hard and elastic. The color varies from a reddish gray to a gray or brown. There is always a fibrous capsule which sends septa into the tumor. There are much enlarged veins on the surface, and the bloodvessels are so abundant that a spongelike appearance is shown on the cut surface. They are usually intimately adherent to the common carotid artery and its branches, but in a few cases they could be peeled off easily. It can be said that their histology shows hyperplasia of the cells around the bloodvessels and the name of endothelioma is most generally accepted, since the most accepted embryological view of the origin of the gland at present is endothelial. The connective tissue septa are apt to undergo hyaline degeneration. The tumor cells are cubical, spindle, triangular, or polydehral in shape. Some have found processes from the angular cells, which seemed to be intimately connected with the capillaries. Chromaffin cells are also found, either in the alveoli or in the stroma. Some fuchsinophile cells are described by Keen and Funke.

The tumors are benign in the early stages; later they grow very rapidly, and not until then do they usually come to be noticed by surgeons. They have a marked tendency to adhere to the surrounding structures, the pneumogastric, the sympathetic, the hypoglossal, and the lingual nerves. In 1 case the tumor was adherent to the anterior surface of the cervical vertebræ; in 2 it reached the base of the skull; in another it bulged into the pharynx. In 5 cases there was enlargement of the neighboring lymphatic glands; in 1 other metastases in the liver were found postmortem; in another the growth was distinctly malignant, rapid wasting occurred, and the patient died without operation. In 4 cases there was recurrence after operation, probably on account of the incomplete removal of the cells of the gland.

Sclerosis. By this term is meant an increase of connective tissue of the carotid gland. This occurs in old age and in patients with syphilis of the carotid artery, whereby an arteriosclerosis is produced. Schaper found that in old age spontaneous dissolution of the cells and increase in connective tissue and bloodvessels occur. Careful examination of all the specimens I obtained, taking into consideration the age of the individual and the amount of connective tissue and sclerosis in the intima of the corresponding carotid artery, revealed the fact that sclerosis of the gland is always associated with sclerosis of the intima of the carotid artery, and the former is proportional to the latter. The process seems to start from the adventitia of the carotid artery, for the interlobular septa first increase in volume, thickness, and proliferative activity, as shown by round cells, elongated endothelioid cells, spindle-shaped cells, and young strands of fibrous connective tissue. In young individuals the connective tissue is very slight in amount, the cell balls seem to fuse with each other, and even the lobules are not very well defined. As

age, or, what is similar, arteriosclerosis, advances, proliferative changes take place, and the interlobular and intralobular connective tissue becomes more evident, separating the elements of the gland into lobules and cell balls; when arteriosclerosis becomes more marked the connective-tissue elements preponderate, the blood-vessels of the interlobular septa become sclerosed, the cell balls atrophied, and eventually the connective tissue, becoming denser and denser, undergoes hyaline degeneration.

Arteriosclerosis produced by syphilis in one case offers an instructive picture.

CASE XXXIII.—Male, aged forty-six years.

Anatomical Diagnosis. Aortic insufficiency and syphilitic aortitis.

Gross Description of the Gland. Only the carotid gland of the right side was obtained. It is made up of fairly distinct lobes, connected with each other by a fibrous vascular band. The larger one is on the posterior surface of the bifurcation, size 8 by 5 by 4 mm., a great deal larger than normal; smaller one located posterior to the internal carotid artery; size 3 by 2 by 4 mm. The arteries are thickened, and patchy calcification of the intima is present. Careful search for the opening of the artery into the carotid body failed to locate it.

Histological Description. The majority of the parenchyma cells are of the small type, with densely staining nuclei. A very striking amount of proliferative changes in the connective tissue, which is very cellular (lymphoid cells, endothelioid cells, and spindle-shaped cells) is present. The cell balls are well bounded by fibrocellular connective tissue. The cells of the stroma are increased, and in fact they seem to predominate, in the cell balls. The bloodvessels are not thickened, on the contrary, they seemed to be thinned by the great bulk of blood that they contain. As the opening of the main bloodvessel of the carotid gland is obliterated and no necrosis of the gland followed, it may be concluded that a good anastomosis with the vasa vasorum took place.

Cloudy Swelling. It was expected that the carotid gland would be affected by toxins and bacteria in the blood. I examined 11 cases of acute and chronic nephritis, 2 of septicemia, 1 of typhoid, 7 of pneumonia, and 2 monkeys (obtained from Dr. H. T. Ricketts) which died of Rocky Mountain Spotted Fever. These infectious and toxic processes, taken as a whole, had very little effect on the carotid gland. Only in one case was a marked change observed.

CASE L.—Male, aged sixty-four years; autopsy twelve hours after death.

Anatomical Diagnosis. Lobar pneumonia.

Gross Description. Only the right side was examined. Gland was about 2 mm. in diameter, placed posteriorly on the bifurcation. It looked small and sclerosed.

Histological Appearance. Gland sclerosed. The parenchyma under low power shows indefinite, dark-staining patches; with high power the parenchyma cells are seen to be somewhat swollen, more distinctly granular, boundaries altogether obliterated, fusing into one degenerated mass with scattered dark nuclei. This is cloudy swelling, but it does not extend to all the cell balls.

In several cases of septicemia the blood was laked, showing the effect of infection, and yet the cells of the gland seem to have altogether escaped pathological alteration.

Hyaline Degeneration. In tumors, hyaline degeneration of the connective tissue has been described by Keen and Funke, Monckeberg, Paltauf, and Kopfstein,[21] due probably to fusion of the fibers and subsequent homogeneous appearance.

As the gland grows older, the connective tissue increases in volume and density, and eventually becomes homogeneous in appearance, and hyaline degeneration takes place. This was particularly marked in Case XLIV (a male, aged forty-five years, dead of septicemia) in which the connective tissue made up half of the volume of the gland.

Other Changes. In Case XXXVII, a male, aged fifty-seven years, dead of chronic nephritis, there was found one very curious lobule. This was choked full of lymphoid cells, which were rounded in shape, poor in protoplasm, with dark, rounded nuclei without visible nucleoli. These must have sprung from the preëxisting connective tissue cells and from the blood stream, since neighboring bloodvessels showed slight increase of lymphocytes. In this particular lobule there were a few parenchymatous cells scattered here and there, as though the round cells had grown in among them and pressed them apart.

Four cases of miliary tuberculosis, and 4 of other kinds of tuberculosis were examined. In one case, a male of about twenty to twenty-five years of age, the tuberculosis was so generalized that even the intima of the arteries seemed to be riddled with millet seed tubercles; and yet in this case, as well as in all the rest, the carotid gland escaped infection.

Examination of glands from the following showed no pathological alteration: One case of multiple myeloma involving the frontal lobe of the brain, 1 case of pseudoleukemia, 1 of glioma of the brain, 1 of glioma of the retina, 4 of heart disease, 1 of gas poisoning, 1 of unknown poisoning, 1 of aortic aneurysm, 3 of cerebral hemorrhage.

Postmortem Changes. Schaper says that the cells contain a delicate hyaloplasm, and it is hard to preserve them true to life. This is the experience of all subsequent investigators. Comparing the animal material which was fresh, and the human material which was

[21] See Keen and Funke

obtained a varying length of time after death, I came to the conclusion that the granules of the protoplasm in the latter seem to dissolve at the periphery, and consequently they are relatively more granular toward the centre around the nucleus, and poorly defined vacuoles are formed apparently by dissolution of these granules. With longer standing, the protoplasm stains very poorly, the edges being ragged in appearance. I had cases examined up to six days postmortem, and the cells do not seem to dissolve entirely, as in the case of the medullary substance of the suprarenal capsule.

SUMMARY. 1. The carotid gland is variable in size, shape, position, and number; it is not always present, or at least cannot always be found in its usual location, and may be absent on both sides or only on one side.

2. It is probably present in all mammals, and is more of a parenchymatous character and more intimately embedded in the adventitia of the carotid arteries in the lower animals than in man.

3. It increases in size as the indvidual grows, because of increase of connective tissue, bloodvessels, and parenchyma. Reaching a certain stage, between twenty and thirty years, it remains stationary for a time, and then only the connective tissue increases, the interlobular bloodvessels thicken, and sclerosis and atrophy of the gland result.

4. There are two types of parenchyma cells, differing mainly in the amount of chromatin and the size of the nucleus. The parenchyma cells of lower mammals are only of one type, which is more similar to the type in man which is characterized by a large rounded vesicular nucleus.

5. The sclerosis of the gland is proportional to the sclerosis of the intima of the carotid bloodvessels, whether due to age or to syphilitic endarteritis.

6. The gland is not very much affected by systemic diseases, the only changes found being one case of cloudy swelling, one of lymphoid infiltration, and frequently hyaline degeneration of the sclerosed stroma in old age.

7. The cells undergo postmortem changes readily, but do not dissolve entirely, as do the cells of the medulla of the suprarenal.

8. The nature of my material was such that no conclusions can be drawn concerning the chromaffin cells.

This work has been suggested by and done under the supervision of Dr. H. Gideon Wells. Drs. Le Count and Bassoe and Mr. Rosenberg have provided facilities for the obtaining of material. Dr. Warren H. Hunter kindly allowed me to get material from his numerous autopsies. To all of them I wish to express my deepest gratitude.

CONGENITAL UNILATERAL ABSENCE OF THE UROGENITAL SYSTEM AND ITS RELATION TO THE DEVELOPMENT OF THE WOLFFIAN AND MUELLERIAN DUCTS.

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The occurrence of congenital absence of the urogenital system is not so uncommon as to detract from the interest of such a case. Although instances have been mentioned by Aristotle, Vesalius (1543), Lopez (1564), Columbus (1572), Bauhinus (1621), Laurentius (1628), Duretus (1635), Riolanus (1648), and Eustachius (1707), they are quite uncertain, as no distinction has been drawn between atrophied, fused, and undeveloped kidneys.

Mosler[1] is among the first of the modern writers to take up this subject in a systematic manner, and he has tabulated 14 cases of absence of the kidney; in 7 of these the ureter had failed to develop, while in 2 the kidney was but rudimentary.

Beumer[2] collected 48 cases between the years 1853 and 1878, and in 44 of these the kidney was completely absent, while in 4 it was rudimentary. He found that genital defects were present in 13, 8 females and 5 males. In 3 of the males the seminal vesicles and vas were both absent, and in 1 the testicles also; in the other the normal genitals were present, but in addition there existed a 38 mm. vagina and a 25 mm. uterus, on the right side. In the females the malformations of the genitals consisted chiefly of bicornate uterus, and absence of one tube or one-half of the uterus or vagina. The ovaries were present in all cases. Schwengers,[3] in 1881, mentions a case of absence of the left kidney and ureter, and Thibierge,[4] the absence of the right kidney and ureter in a boy aged nine and a half years. Greenfield[5] describes 2 cases of absence of kidneys and ureter in 1 of which there was also absence of the testicle, vas, and vesicles on the affected side. Mention may also be made of Murchison's[6] case of absence of the left kidney, Hillier's[7] left kidney, Bruce's[8] left kidney and ureter, and both Kelly's[9] and Duckworth's[10] left kidney. Coen,[11] Guttmann,[12] Bartscher,[13] Ogsten,[14] and Gatti[15] report similar cases. Lamiere[16] cites the absence of a kidney in an

[1] Arch. der. Heilk., Jahr., 1863, iv, p. 289.
[2] Virch. Arch., 1878, lxxii.
[3] Berl. klin. Woch., 1881, p. 481.
[4] Prog. med., vol. x, p. 656.
[5] Trans. Path. Soc. London, 1884, vol. xxviii.
[6] Ibid., vol. x, p. 190.
[7] Ibid., vol. xv, p. 46.
[8] Ibid., vol. xvii, p. 175.
[9] Ibid., vol. xix, p. 274.
[10] Trans. Lond. Path. Soc., vol. xx.
[11] Vide Ballowitz, 22.
[12] Arch. f. path. Anat., 1883, vol. xcii.
[13] Vide Brown, 21.
[14] Obst. Soc. Lond., 1880, vol. xxi, p. 57.
[15] Cited by Brown, 21.
[16] Jour. de Sci. Méd. de Lille, 1892.

individual aged sixty-four years. Coen found, in a study of 33 monstrosities, that the kidney was absent in 15 and the adrenals were larger than normal. Gubbins[17] reports the absence of the left kidney, ureter, and adrenal, and Mackey[18] a single (right) kidney in a child aged two years and nine months. Eschaquet[19] describes the non-development of the left kidney, ureter, ovary, and tube, and one-half of the uterus. Rayer[20] cites 17 cases and in 12 of these the ureters and vessels were not present. Tweeding[21] describes the absence of the right kidney, ureter, and vessels in a woman aged thirty years, while M. Brown[22] reports 3 cases in 12,000 autopsies.

Ballowitz[23] was the next writer to collect cases of this condition. Up to 1895 he computes 213 cases, and states that there are probably 25 more, which represent literature to which he had not access. I have been able to trace some of the latter cases, and the results will be noted later. Ballowitz describes 3 cases of his own. In the first there was no trace of the left kidney, ureter, or vessels, but the adrenal was present; the left vas, seminal vesicles, and ejaculatory duct were missing and the testicle atrophic. In the second the right kidney was wanting, and in addition the ureter and vessels; the genitals, however, were normal. In the third the left kidney, ureter, vas, and ejaculatory duct were absent, and the testicle soft and smaller than usual.

From a study of the 213 cases Ballowitz found that the left side was more commonly affected than the right, in the proportion of 117 to 88, with 8 undifferentiated. This condition occurred more often in the male than in the female, and here on the left side oftener than on the right, 70 to 42. In the female the proportion was nearly even. Usually rudimentary vessels were found, and in all but 15 the ureters were undeveloped. There was the usual compensatory hypertrophy of the remaining kidney. Of the 213 cases, in only 103 was there any mention made of the genitals. In only 73 were there any defects; 41 occurred in the females and 28 in the males; in 4 the sex was not mentioned. The efferent system, derivatives of the Wolffian and Muellerian ducts, seemed more commonly affected while the ovaries and testicles showed little alteration.

In 18 the uterus was bicornate (10 right and 8 left); in 5 the uterus and tubes were both absent; in 10 the uterus was bicornate and contained a double cavity, while in 2 a double vagina existed. In 3 cases alone were the ovaries on the affected side absent. The external genitals were seldom altered.

In the male the vas and seminal vesicles were usually affected. Both were absent in 13, while in 2 the vas was wanting and the

[17] Brit. med. Jour., January, 1883, p. 115.
[18] Ibid., September, 1887, p. 626.
[19] Prog. med., Paris, 1875.
[20] Traité des Mal. des Reins, 1841.
[21] Jour. Anat. and Physiol., 1893-94, vol. xxviii.
[22] Ibid., p. 194.
[23] Virch. Arch., 1895, vol. cxli, p. 309.

vesicles atrophic; in 3 the vesicles alone were absent, while in only 4 was the ejaculatory duct missing. In 2 the testicles were entirely wanting, while in 8 they were small and atrophic.

The following are among the doubtful cases of Ballowitz, and these have been confirmed by the writer. Darby[24] found an absence of the left kidney and ureter; Thatcher[25] mentions 2 cases of absence of the kidney and ureter, but makes no mention of the genital defects, sex, or side affected; in the same meeting Armstrong[26] stated that he had seen such a defect in a man. Peabody[27] cites 2 cases, 1 of the right kidney and ureter and the other of the left kidney and ureter and the presence of a uterus bicornis. Isaacs[28] noted the absence of the left kidney and ureter in a child of three months; Polk[29] found the right kidney missing, postmortem, in an individual in whom the left had been removed for disease; in addition he found the right ureter, vagina, and uterus absent, but both ovaries were present. Holt's[30] case was that of a rudimentary, cystic kidney, and therefore cannot be counted. Wier[31] noted the absence of the left kidney and ureter, and Church[32] mentions the same defect in a female, and states that he had seen another such case at an autopsy. Ingals[33] found the right kidney wanting in a female, while Gouley[34] noted a single pelvic kidney, apparently the right, that received its blood from the middle sacral artery. Cargill[35] merely mentions the absence of the left kidney in a male, while Hutchinson's[36] case represents a cystic kidney. Several references given by Ballowitz were incorrect, and the remainder could not be obtained.

In addition to the above, I should like to note some cases overlooked by Ballowitz. Tweeding's case was one of absence of right kidney, ureter, and vessels in a woman aged thirty years; Dr. Fenby[37] and Dr. Walker mentioned a case in which no trace of either right kidney or ureter could be found, while Dr. Falk[38] cites one in which there was absence of the right kidney and ureter. Prudden,[39] in his discussion of Holt's case, says that all cases that he had reported showed absence of the left kidney. Church[40] mentions 2, while Ballowitz gives him credit for only 1. Since 1895 Newman[41] reported absence of left kidney, ureter, and vessels; Sunderland[42]

[24] Proc. Phila. Path. Soc., 1857 to 1860, p. 199.
[25] New York Med. Record, 1892, vol. xlii, p. 517.
[26] Ibid., vol. xlii.
[27] Bull. New York Path. Soc., 1881, p. 138; New York Med. Record, 1882, vol. xxii.
[28] New York Jour. of Med. and Collat. Sci., 1858, p. 218.
[29] New York Med. Jour., 1883.
[30] New York Med. Jour. and Rec., 1887, vol. xxx, p. 696.
[31] Indian Med. Gaz., 1872, vol. vii, Calcutta.
[32] Jour. Amer. Med. Assoc., 1884.
[33] Chicago Med. Jour. and Examiner, 1875, vol. xxxii.
[34] New York Med. Record, 1872, vol. vii, p. 433.
[35] Prov. Med. Jour. and Retros. of Med. Sci., 1884, vol. ix.
[36] New York Med. Jour. and Rec., 1869.
[37] New York Med. Rec., March, 12, 1881.
[38] Arch. f. path. Anat., 1883, p. 558.
[39] New York Med. Rec., 1886, p. 696.
[40] Jour. Amer. Med. Assoc., 1884.
[41] Glasgow Hos. Rep., 1898, p. 120.
[42] Glasgow Med. Jour., February, 1898.

and Edington reported 4, Moore 1, with unilateral aplasia of the genitals. He believed that males were affected more commonly than females in the ratio of 2 to 1. Edington[43] reported another, and Glazebrook[44] one in a woman aged thirty years, in conjunction with atrophic adrenal. If we allow 240 cases up to the appearance of Ballowitz's paper, then at least 15 more must be added, making a total of at least 255.

In reference to autopsies, Brown found 3 cases in 12,000, Morris[45] 2 in 8068, Sanzalli[46] 3 in 5348, Menzies[47] 2 in 1790, Rootes[48] 1 in 600, and Ballowitz 1 in 617.

In considering the condition in animals, the following has been found: Botalli[49] and J. Van Horn found a single kidney in a dog, while Stoss[50] found the right kidney and ureter absent in a sheep; Newmann[51] noted the same in a horse, and Matthias[52] in a pig. Reterer[53] and Rogers found an absence of the right kidney and ureter, complicated with genital defects, in a rabbit; the right ovary was present, but the corresponding uterus and vagina were absent, while the tube ended in a nodular swelling. Harrison[54] found a rabbit in which the left kidney was absent, the vagina rudimentary, and the uterus and vagina entirely wanting; the ovaries, however, were present. Sutton[55] states that this malformation occurs in the horse, sheep, pig, and hen, while a student of Brown[56] found the defect in a pigeon. According to Marek,[57] congenital aplasia in animals is not rare. Prettner found at the abbatoir that in 15,000 animals killed, two pigs showed agenesis of the kidney, one right and and one left, and one steer exhibited the same condition.

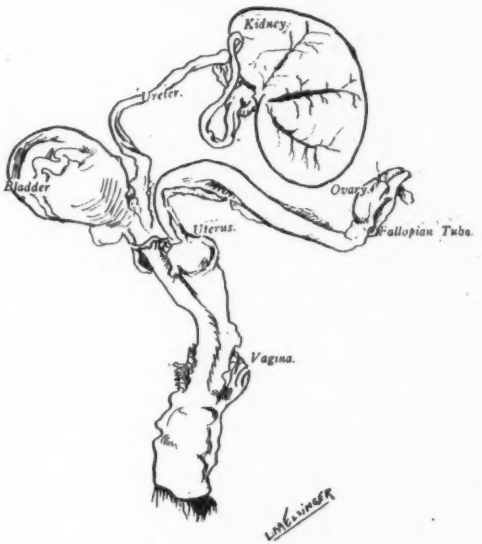
I desire to add another case that occurred in a cat. The organs shown in the accompanying illustration were brought to me by a student who had dissected the cat. As can be seen, there is entire absence of the kidney, ureter, and genital tract of the same side. No trace of a ureteral orifice could be detected in the bladder, nor was there a stump of a ureter. In the genital system the narrow vagina leads into a left uterus only. As to the presence of both ovaries, I cannot say, as the student had not noted those structures. As the ovaries are so rarely absent, it is presumed that both were present. The specimen was mounted in special gelatin, as described by Coplin,[58] and is still as good as when prepared in 1903.

[43] Lond. Jour. of Anat. and Physiol., 1903-04.
[44] New York Med. Jour., 1905, p. 174.
[45] Surgical Dislocations of the Kidney, London, 1885.
[46] Cited by Ballowitz, 22.
[47] Jour. Anat. and Physiol., 1887, vol. xxi.
[48] Lancet, 1866, vol. ii, p. 251.
[49] Quoted by Ballowitz.
[50] Deut. Zeit. f. Tiermed. und vergl. Path., 1886, vol. xii, 284.
[51] Glas. Hos. Rep., 1898-99, p. 131.
[52] Quoted by Brown, 21.
[53] Comp. Rend. de Biol., 1893, Ser. 9, Tome v.
[54] Jour. Anat. and Physiol., 1893-94, vol. xxviii, p. 401.
[55] Cited by Brown, 55.
[56] Jour. Anat. and Physiol., 1893-94, vol. xxviii, p. 194.
[57] Deut. Tier. Woch., Hanover, 1905, vol. xiii.
[58] Jour. Amer. Med. Assoc., August 13, 1905.

In examining cases in which the genital defects are mentioned, the table on p. 116 has been prepared.

In the male we find the vas, seminal vesicles, and the ejacutatory duct most commonly affected, while the testicle seldom shows changes. No case shows complete absence of the internal genitals, including the testicle. In the female the uterus is most commonly affected, while the ovary is rarely missing.

It might be of some interest to consider the embryology of the urogenital system, with the idea of throwing some light on these malformations and the development of the Muellerian duct.



Urogenital system described in the text.

In considering the embryology we must describe the origin of the pronephros, mesonephros, and duct, and the duct of Mueller. In the development of this system the earliest organ to appear is the pronephros. This consists of a longitudinal tubule developed from the mesoderm, opening caudally into the cloaca, and a few transverse tubules at the cephalad end of the duct. The cephalad extremity opens into the abdominal cavity by means of a trumpet-shaped expansion. This pronephros, however, functionates only in the larval stages of amphibians, and gives way to the Wolffian body, or mesonephros. This consists of transverse tubules that connect with the pronephric duct, and the latter then becomes known as the mesonephric, or Wolffian duct. In the lower vertebrates

TABLE SHOWING CONGENITAL DEFECTS OF THE GENITAL AND URINARY SYSTEMS.

Author	Case No.	Sex	Vas	Vesicles	Ejac. duct	Testicle	Tubes	Uterus	Vagina	Ovary	Remarks
Mosler	2	Male	A	—	—	A					
	11	"	—	—	—	⅔ A	....	....	....	....	Representing 2 cases
Beumer . . .	4	Male	A	A	—	Small					
	12	"	—	A	—	—					
	14	"	A	—	—	A					
	29	"	—	—	—	Rudimentary uterus and vagina					
	48	"	Slight	Slight	—						
	35	Female	..	....	..	....	....	½ A	½ A	Small	
	36	"	..	....	..	....	....	Bicornate	½ and blind		
	37	"	..	....	..	....	....	"	Vagina duplex		
	39	"	..	....	..	....	....	Duplex	Vagina simplex		
	41	"	..	....	..	....	....	Left horn absent	—		
	42	"	..	....	..	....	....	Left horn absent	—		
	43	"	..	....	..	....	....	Unicornis	—		
	45	"	..	....	..	....	....	Bicornis	—	....	Representing 13 cases
Ballowitz . . .		Male	A	A in 3							
		"	A	Atrophic in 2							
		"	—	—	A in 3						
		"	—	—	A in 4						
						A in 2					
						Small and atrophic in 8	....	....	....	....	Representing 41 males
		Female	..	....	..	.. .	....	Bicornate in 18 (8 l. and 10 r.)			
								Double cavity in 10			
							Absent in 5	Absent in 5.	Double in 2		
							Absent in 3	Absent in 1	Absent in 1	Absent in 3	Representing 28 females
Peabody . . .		Female	..	....	..	....	....	Bicornis			
Polk.		"	..	....	..	....	....	A	A		
Ballowitz . . .		Male	A	A	A	Atrophic					
			A	—	A	A					
Moore		Aplasia of the genitals.									
Greenfield . . .		Male	A	A	—	A					
Eschaquet . . .		Female	..	....	..	....	A	Unicornis	—	Absent	
											Total males 49 Total females 41

A = absent. — = nothing stated in regard to those organs.

(selachians), Semper, Balfour, Hoffman, and Rabl believe that the Muellerian duct is derived from the mesonephric duct by segmentation, or longitudinal splitting of this duct; the trumpet-shaped upper extremity of the mesonephric duct falls to the Muellerian tube in this division. This occurs also in amphibians, according to Hoffmann and Furbringer, with the exception that the fimbriated end is derived especially from the abdominal mesothelium by a thickening and latter invagination of the mesothelial cells; this part finally gains connection with that derived from the mesonephric duct. Semon, however, finds that this does not occur in all amphibians. In reptiles, birds, and mammals the origin of the Muellerian duct is in dispute. Waldeyer, Braun, Gasser, Janhosik, Mihalkovics, and others do not believe in the segmentation, or splitting, of the Wolffian duct as the origin of the Muellerian tube. This seems corroborated by Wiedersheim, Hoffmann, and Nagel, as they find that it appears later than the pronephric duct and is composed of a solid cylinder of cells with a trumpet-shaped invagination of the mesothelium of the body cavity, this latter becoming the ostium abdominale of the oviduct.

The mesonephric duct opens caudally into the cloaca. From this end the metanephric evagination, which becomes the permanent kidney, ureter, and pelvis, appears. In addition to this, in the male this duct gives rise to the body and globus minor of the epididymis, the vas, seminal vesicles, and ejaculatory duct, but nothing of importance in the female. In the female the ducts of Mueller fuse for over half of their length, and ultimately form the uterus and vagina, while the unfused portions constitute the oviducts.

If we examine the table, we note that the genitals are defective in 90, 49 males and 41 females, and one doubtful case of Moore's. In all there are about 255 cases of absence of the kidney and, of these, 100 show decided defects in the genitalia, over one-half of which occur in males. The parts affected are as follows: The vas practically absent in 22, the vesicles in 20, the ejaculatory duct in only 9, and the testicles in 15. In the female we find the uterus entirely absent or bicornate in 12, the vagina absent or reduced one-half in only 5, and the oviducts missing in 9, while the ovary was absent in but 4.

Absence of the kidney may be due to the following causes: (1) Failure of the metanephric evagination to appear, even though the mesonephric duct and body be perfect; (2) appearance and early retrogression of the metanephric evagination; (3) failure of the pronephros, and therefore also the mesonephros, to appear.

In the first instance the absence of the kidney in the male would naturally be attended by few if any genital defects, and these would be coincidental and not sequential. In the second instance the resultant conditions would be the same. In the third all such cases would be attended by absence of the kidney, ureter, efferent ductular

system of the testicle, vas, and vesicle on the affected side. From the irregularity of the occurrence of the genital defects, the absence of the kidney seems to be due entirely to the first or second cause; the genital defects then occur secondarily, and not as a direct result of absence of the kidney; that is, they are merely coincidental. Another reason for taking this view is the fact that in most of the cases of absent kidney and ureter the vessels also were absent or rudimentary, pointing more to the first or second cause.

In the female in the first or second cause there would be no genital defects, whether we assume that the Muellerian ducts are derived from the mesonephric duct or independent; such defects would then be merely coincidental. In the third cause, if the Muellerian ducts were derived from mesonephros partially or entirely, then as a result of its absence the entire internal genitals should be practically absent. In fact, that has not occurred in any case. In reality, the uterus was absent or bicornate in but one-third of the cases. The facts as found in the adult body seem to indicate that the Muellerian ducts have an independent origin and are not derived from the mesonephric duct by segmentation, at least not in the higher vertebrates.

REVIEWS.

SYPHILIS: A TREATISE FOR PRACTITIONERS. By EDWARD L. KEYES, JR., M.D. Pp. 577; 69 illustrations; 9 plates. New York and London: D. Appleton & Co., 1908.

THE "foreword" to this book, written by the father of the author, states that "the facts upon which the volume rests are the classified cases" from private office books kept during forty years, other and similar work by the author himself, and "gleanings" from Dr. Van Buren's case books. Dr. Keyes, the elder, speaks of himself as "the godfather of many of the generalizations advanced," but adds later that they "corroborate quite uniformly similar generalizations of other authors often based upon statistics numerically superior." This would appear to be correct, and, so far as the views advanced are those generally accepted by syphilographers, there is no occasion to review them in detail. As a rule, they are well and correctly stated.

Even in a scientific field so thoroughly cultivated as that of syphilis there must, however, be outcroppings of new beliefs or new theories, which will seem to one observer firmly rooted and valuable, to another ephemeral and useless, or harmful; and there are many more that at any given time will be regarded by some as having demonstrated their value, but by others are thought still to be on trial. Here and there in this treatise are to be found teachings that seem to the reviewer too dogmatic for the present state of our knowledge, and generalizations that are not yet justified by the facts at our command.

It is stated that the advocate of the regulation of prostitution can show, as a result of regulation, "no marked decrease in the incidence of venereal disease." This is too sweeping. Diminution of such diseases has repeatedly followed what the author calls "reglementation," and, indeed, in a foot-note two pages farther on, he says: "The incidence of syphilis is highest in the English army (75 per mille per annum). Then follow the United States (33.98), Austria (19.2), Russia (12.8), France (6.7), Holland (4.9), Bavaria (4.3), Prussia (4.0). The prudish Anglo-Saxon refusal to countenance protective measures for the health of the army here shows its results." This does not refer exclusively to the control of prostitution, but it, of necessity, includes it.

The assertion that the mother of a syphilitic child "almost invariably ultimately breaks out with tertiary syphilis," and the statement that "all syphilis is relapsing syphilis," are not in accord with the observed facts. Such a mother may develop tertiaries; any case of syphilis may relapse. These are facts, and, indeed, the context shows that, in the latter instance, this is what was meant; but accuracy has been sacrificed to terseness and to a certain striving for epigrammatic statement noticeable throughout, which tends to make agreeable reading but unsound teaching.

We believe, with the author, that the "spirocheta of Schaudinn is either the cause of syphilis or one phase in the life cycle of some microörganism which is the cause of syphilis," but we would not yet venture to say that it "seems certain," nor can certainty be asserted until the organism has been successfully cultivated. The results of experimental inoculation upon the anthropoid and other monkeys are now adding constantly to our knowledge of the disease, but they do not yet justify the absolutely unqualified announcement—italicized—that "syphilitic secretions cease to be infectious after twelve to twenty-four hours, and much sooner (at most six hours) when dry." This may be true, but until we *know* that it is true the practical possibilities of evil from accepting and acting upon it should not be forgotten.

Even more important is it to remember the present lack of demonstrative proof of the following statement: "The only personal prophylaxis against syphilitic infection lies not in cauterization or excision, but in inunction with a strong mercurial ointment within one hour of inoculation." The author adds that "perhaps as late as six hours the remedy might still be efficacious." It is harmless, and may well be tried, but dependence on its efficacy at any time might certainly lead to disastrous consequences. It is to be feared that extended trial will show that (as in the local treatment of chancre and other open lesions) mercury may have some slight local effect, but that—as the author says elsewhere—systemic treatment must be employed to obtain "real results." He adds: "Experiment has proved that inunction or injection of mercury encourages healing of adjacent syphilitic lesions; but clinically this local action is of little importance."

It is impressive to say, "Nothing is certain in syphilis except its uncertainty," but, as a matter of fact, it is one of the most orderly of the infectious diseases from its incubation to the termination of the period of clinical contagion.

It is open to reasonable doubt that "syphilis in woman, though it ceases to be transmissible by contact quite as early as in man, may continue transmissible by heredity for an indefinite number of years." At any rate, the statement should be more closely coupled with the undoubted rule—accurately formulated a few pages later—that "the probability of syphilitic heredity, like the probability

of contact infection (in matrimony), is overwhelming in the first year of the disease, great in the second and third years, slight in the fourth and fifth years, and negligible thereafter."

The chapter on the treatment of syphilis, and that on the physiological and toxic effects of mercury, deserve almost unreserved commendation. In that on the administration of the drug, the advantages of insoluble injections appear to the reviewer to be overstated and the dangers and disadvantages correspondingly minimized. Embolism, of which a number of fatal cases—not "one or two"—have been reported, is scarcely to be called a "petty inconvenience," even in comparison with the benefits sometimes unquestionably to be derived from the injection method. Nor do inunction and internal administration, in the opinion of many syphilographers, rank in efficacy below fumigation, as is asserted by the author.

It is true that the characteristic early syphilitic adenitis may not occur so that the nodes are palpable, but such cases are so rare that the statement that if the nodes are not enlarged "the evidence has no value" would be most misleading if accepted as a diagnostic rule. The chapters on regional syphilis are valuable summaries of our knowledge of the subject, those on special organs or systems having been reviewed for the author by various specialists, to whom due acknowledgment has been made. No mention of mammary syphilis was found by the reviewer, except in relation to chancre of the nipple.

Here and there a little carelessness in writing—or in proofreading—is manifest, *e. g.*, "among the literature;" "this . . . spread John Hunter's name," "a host of practical queries . . . are constantly clamoring for answer;" and one remarkable paragraph, which reads: "Thus Fournier narrates cases of infection transmitted to a child by its uncle, who blew his syphilitic child; and this exception is fully compensated for by 3 other case in which a kindly lady bound up the scratched knee of a little child with her handkerchief saturated with saliva!" The "kindly lady" reminds one of the sort of redundancy recently exemplified by a critic of English, who says that it is as though one should breathlessly announce: "A gentleman of much personal refinement has just fallen from the third story window," or "A lady with a fine command of modern languages is struggling for her life in the river."

To emphasize such criticism would, however, be not only ill-natured, but unfair to a book which shows industry, and experience on the part of its author. As the most recent addition to the treatises on syphilis, it is deserving of careful reading, and, as a rule, in its broader teachings may safely and advantageously be followed by the general practitioner. J. W. W.

MODERN SURGERY. By JOHN CHALMERS DA COSTA, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College, Philadelphia. Fifth edition; pp. 1283; 872 illustrations. Philadelphia and London: W. B. Saunders Co., 1907.

THIS volume is the fifth edition of Da Costa's *Surgery*, and a glance at the copyright page shows that in addition the book has been reprinted many times since its original appearance in 1894. With such a history, praise seems superfluous and criticism needless. Many of the sections have been corrected and much new matter has been introduced, particularly along the lines of improved technique. The encyclopedic character of the book is impressed upon one before many pages have been turned, and the author is particularly to be commended for the frequent interpellation of references, which not only serve to show the practising physician where such information was acquired, but also tends to encourage the undergraduate student to read further than his text-book. Almost everything relating to surgery, whether in pathology, diagnosis, or operative technique, seems to be included, and it is this jumbling of the good with the bad, the modern with the old, that offers a point for criticism. Many methods are mentioned and some are described in detail, only to have added to them at the end that the author does not use them in his own practice, or that they have been generally discarded by the profession. The earlier chapters devoted to surgical pathology are generally excellent, although most of the illustrations are old and give but little idea of the lesions they are meant to illustrate. The chapters upon the treatment of hemorrhage, upon tumors, fractures, brain injuries and tumors, and spinal cord injuries, are particularly good. In the treatment of the latter the advocacy of early operation in all cases in which the symptoms are significant is based on sound reasoning. The surgery of the respiratory and the genito-urinary organs is well balanced and generally excellent. In discussing the treatment of acute peritonitis the author gives a splendid presentation of modern methods, in particular condemning flushing of the abdominal cavity. The Fowler position and Murphy's method of proctolysis are advocated. The entire section devoted to the various diseases and injuries of the abdomen is most interesting and clear cut. The chapter upon the thyroid gland is also well arranged; the author prefers local anesthesia for thyroidectomy and makes no mention of the parathyroids in relation to tetany.

In the chapter upon inflammatory diseases of bone, caries, necrosis, osteitis, and chronic osteomyelitis are not discussed with the usual clearness characteristic of the book. As the author states, "the term caries is seldom used today except loosely," and the same might have been said of necrosis. Descriptions of the sequential lesions of osteoperiostitis or of osteomyelitis, with appropriate

treatment of caries or necrosis as they occur, would seem the preferable way of teaching, instead of placing the latter terms in heavy type as separate diseases. Bier's method of passive congestion is barely mentioned, and that only in tuberculosis, in which, except in early cases, it has its most limited field. The method of treatment recommended for what is called "osteo-arthritis" is inadequate, no distinction being made between the atrophic and the hypertrophic varieties. Objection might also be taken to the statement that removal of the Gasserian ganglion is dangerous, bloody, and difficult; if the patient is operated on in the sitting position, and if the skull opening is made posterior to that of the Hartley-Krause method, the objections disappear, and a mortality of 10 per cent. is not particularly objectionable when we consider the life of torture which such patients must lead. The pathology of cysts and tumors of the mammary gland, as given, is very confusing, and a scirrhus cancer of the breast is not "almost as hard as stone."

These few criticisms are not made with a view of minimizing the value of this book, because, as it stands, it is probably the best one volume text-book on surgery that we have. The different sections are particularly well balanced, lengthy and complete descriptions being given of important subjects and diseases especially common in surgical practice, and yet the rare and unimportant lesions are adequately described. The 1200 pages represent an immense amount of labor in condensation, and while the omissions mentioned have been noted in glancing through the book, yet a similar list could be found in any other text-book. The index is very complete.

G. P. M.

SURGICAL EMERGENCIES. By PERCY SARGENT, M.A., M.B., B.C. (Cantab.), F.R.C.S., Surgeon to Out-patients, St. Thomas' Hospital, London. Pp. 256. London: Henry Frowde, Oxford University Press; Hodder & Stoughton, 1907.

THIS book was written for those who "require in any emergency, not academic discussions concerning the relative value of different methods, but explicit directions as to some definite line of treatment, which, if not always the best, has, at least, the merit of having been found by experience to be sufficiently good." With this gentle apology in his preface, the author introduces us to an eminently practical work, in which he succinctly describes the symptoms and treatment of such surgical emergencies as hemorrhage, burns, fractures, acute infective diseases (including those of the abdomen); injuries of the abdomen, strangulated hernia, respiratory obstruction; as well as emergencies connected with the urinary system;

injuries of the neck, chest, nervous system, ear, and eye. It is a volume which will prove of interest to every active surgeon, and which, better than any handbook with which we are acquainted, will fill the want of resident physicians on duty in the receiving wards of large hospitals.

There can be few criticisms of the treatment recommended; it is, with few exceptions, that which is approved by judicious surgeons the world over as expedient and safe. Yet not to even mention elevation of the limb to control hemorrhage seems an unpardonable omission; and we think the routine use of general anesthesia in the reduction of fractures is needlessly commended. The treatment advised for tetanus seems scarcely up to date, and we are surprised to be told that the majority of surgeons have abandoned intubation in diphtheria in favor of tracheotomy. Nor can we agree with the recommendation to sponge away all peritoneal exudate in cases of diffuse peritonitis, and to administer calomel and salines as soon as the patient is able to swallow.

On the whole, the book admirably fulfils its purpose, and the points adversely criticised have been noted merely because we had thought they were no longer mere matters of opinion, but of fact.

A. P. C. A.

ATLAS AND EPITOME OF DISEASES OF CHILDREN. By DR. R. HECKER and DR. J. TRUMPP, of the University of Munich. Authorized translation from the German. Edited by ISAAC A. ABT, M.D., Assistant Professor of Diseases of Children in the Rush Medical College, Chicago. 48 colored plates and 147 black and white illustrations. Philadelphia and London: W. B. Saunders Co., 1907.

THIS latest volume of the well-known *Lehmann's Medical Hand-Atlases* is a worthy companion to the various volumes already translated. Like them it is especially noteworthy for the profusion and beauty of its illustrations, the large number of colored lithographs presenting most striking and lifelike reproductions of many eruptive diseases, those picturing the clinical appearances of the exanthemata being especially successful.

While the volume is essentially a picture-book, the text is by no means a mere descriptive narrative to fit the illustrations, and its value in the original has been greatly enhanced by Dr. Abt's judicious editorial annotations. Numerous changes in the text have been made by the American editor in the sections on therapeutics, necessitated by the differences in treatment obtaining in this country, and by the fact that many of the special food preparations and various forms of apparatus used in Germany are either unknown or unobtainable here.

It is not expected that the hand atlas should take the place of larger and more comprehensive text-books on diseases of children. To the most important subject of artificial feeding, for instance, only four pages are devoted, and nearly two pages of this allowance are given to an enumeration of patent foods and the special milk products which are so generally used in Germany. As a supplement, however, to clinical study, within the reach of every student, the present volume should be a welcome addition to any working library. T. S. W.

MOSQUITO LIFE. By EVELYN GROESBEECK MITCHELL, A.B., M.S. Pp. 281. New York: G. P. Putnam's Sons, 1907.

THE American people, especially those living in our larger cities, have shown distinct apathy in regard to great hygienic problems, in spite of thousands of preventable deaths occurring yearly from communicable diseases. There seems to be but little desire to expend money to wipe out endemic diseases, such as typhoid fever and tuberculosis. Any book, therefore, that may arouse public interest in preventive medicine, and that shows reasons for and means of checking disease has, in this country, a distinct value, even though the story may not be new, and even though it serves merely as a reminder of well-known facts.

The volume by Evelyn Groesbeeck Mitchell falls into this class of books. Mosquitoes are discussed from various points of view. "How and where they breed, how they bite, how they transmit disease, how long and on what they live, how they may be identified in their various stages, and finally, but not the least important, how they may be locally controlled." The book contains on all these subjects many original observations made by the author and by Dr. James William Dupree, a life-long student of the subject. The death of Dr. Dupree prevented him from bringing forth his own observations, but the author has done this, together with much praise and esteem for the man and his work.

The book is written in a semi-technical style, and requires no extensive zoölogical training for its successful use in the field and in the laboratory as a means of identification of the various species and for much information as to their habits. The keys for identification are especially good features of the book. The literary style is somewhat unfortunate. It neither has the seriousness of a thoughtful scientific treatise, nor does it contain enough true literary merit to make it a book to which one would likely turn for diversion. The author includes a number of trivial things that annoy when the book is put to serious scientific use.

The chapters on malaria and on yellow fever and other diseases

are largely taken from the notes of Dupree. They contain nothing that is especially new to medical science. Some of the statements are not in accord with the ideas usually held by the profession, and one or two rather obvious errors can be found. However, the book should be of distinct value as a guide for determining the various species and as an authority on their habits and their dangers, information that should be sought for in all mosquito-infested districts before attempts at control of the pests can be intelligently undertaken. G. C. R.

INSANITY AND ALLIED NEUROSES. A PRACTICAL AND CLINICAL MANUAL. By GEORGE H. SAVAGE, M.D., F.R.C.P., with the assistance of EDWIN GOODALL, M.D. (Lond.), B.S., F.R.C.P. New and enlarged edition; 6 colored plates and 45 illustrations in the text. Chicago: W. T. Keener & Co., 1907.

THE fourth edition of a manual on this subject does not deserve an extensive review. This edition does not differ essentially, except in the matter of the illustrations, from the previous third edition. With 45 illustrative cuts in the text and 6 colored plates, the book may be considered to be fully illustrated; the new cuts have added to the value of the work. The only criticism to be advanced against this book is the fault of ultra conservatism. Practically none of the newer ideas concerning the insanities of adolescence are included. The same conservatism is seen in the general classification of the insanities. The chapter on the commitment of the insane follows the British Law, and, of course, will be of relatively little value to the American student and practitioner. The book can be recommended as a good, conservative presentation of the subject of insanity, for students and practitioners. D. J. McC.

A TEXT-BOOK OF PRACTICAL GYNECOLOGY. By D. TOD GILLIAM, M.D., Emeritus Professor of Gynecology in Sterling (Ohio) Medical College. Second Edition; pp. 621; 364 illustrations. Philadelphia: F. A. Davis Co., 1907.

As stated in the preface to the earlier editions, Dr. Gilliam's book has been prepared to meet the demands of the student and the general practitioner. This it does in a very satisfactory manner. In this second edition the book has been brought well up to date by the inclusion of new material. The whole field of gynecology proper is embraced, and, in addition, chapters are included upon diseases of the rectum and of the kidneys. The operation devised by the author, and known by his name, for the correction of retrodisplace-

ments of the uterus, is well and fully described. It has been tried and found successful by the technique of the author, and also by many other surgeons, with slight modifications, and is, we believe, the best of the round ligament operations. We would suggest, however, that the description of the operation of ventrosuspension be altered in the next edition of the book, since, for those who are still advocates of this form of intervention, the present description is not adequate. We refer to the statement that as a means of uterine support the peritoneum is alone depended upon, to the exclusion of small portions of the recti muscles. We note with pleasure the general definiteness of statement throughout the book, as, for instance, the author's absolutism in his statement that it would be well if all women between the ages of thirty-five and sixty years could be looked after by a competent gynecologist, and, when considering the use of the uterine curette, that "it is a question, indeed, if the curette used postpartum has not killed more than it has saved." The profession in general needs clear, concise, and, above all, dogmatic statements such as these. W. R. N.

A History of Nursing. The Evolution of the Methods of Care for the Sick from the Earliest Times to the Foundation of the First English and American Training Schools for Nurses. By M. Adelaide Nutting, R.N., Superintendent of Nurses in the John Hopkins Hospital, Baltimore, and Lavinia L. Dock, R.N., Secretary of the American Federation of Nurses and of the International Council of Nurses. Vol. I, pp. 549, Vol. II, pp. 461; illustrated. New York and London: G. P. Putnam's Sons, 1907.

The two volumes prepared by Miss Nutting and Miss Dock comprise an exceedingly interesting account of nursing from the earliest available records to the time of the development of modern nursing. The work is divided into three parts, of which the first relates to the pre-Christian period and consists of a description of the care of the sick of primitive man and in India, Ceylon, Egypt, Babylon, Assyria, among the Jews, and in Greece and Rome. Part II comprises the period from the first to the close of the eighteenth century; here is well described the leading part in nursing taken by woman with the advent of Christianity, the relationship of the Roman matrons to the early hospitals, the rise, development, and progress of religious nursing orders, and the later development of nursing apart from the influences of the church. Part III deals with the period from the close of the eighteenth century to the development of modern nursing. Much that is of interest in this period naturally centres about

Florence Nightingale, and it is very well told. There is a chapter on the development of nursing in America, and another on a trio of early training schools—that of Bellevue Hospital in New York, that of the Massachusetts General Hospital in Boston, and the Connecticut Training School in New Haven. Throughout both volumes there is ample evidence of an unusual amount of investigation, of the searching of old records, and of the sifting of much data. The result is a credit to the authors and a noteworthy contribution to the literature of nursing; it must prove a source not only of information, but also of delight to all, physician, nurse, and layman, who may have the good fortune to read it. A. K.

THE CLIMATIC TREATMENT OF CHILDREN. By FREDERICK L. WACHENHEIM, M.D., Chief of Clinic, Children's Department, Mount Sinai Hospital Dispensary, New York. Pp. 400; 5 charts. New York: Rebman Company, 1907.

THE common sense which chooses the apparently unoriginal but none the less useful method of "tempering the wind to the shorn lamb" is worth noting in this day of fads; wherefore this book, while intended primarily as a reference book for the pediatrist, should prove agreeable and wholesome reading to overzealous patients or those interested in the care of delicate children. The author has given considerable space to the climatic conditions in North America, with special reference to the value as health resorts of the different parts of the country, and for those patients who must be treated at home there is much good counsel. R. M. G.

DISEASES OF THE SKIN. By HENRY W. STELWAGON, M.D., Ph.D., Professor of Dermatology in the Jefferson Medical College, Philadelphia. Fifth edition; pp. 1150; 301 illustrations. Philadelphia and London: W. B. Saunders Co., 1907.

THE fifth edition of Stelwagon's *Diseases of the Skin* brings to our attention a book that has been five times revised in as many years—than which there can scarcely be better evidence of conspicuous merit. In the present edition the noteworthy changes are in connection with diseases of warm climates, such as frambœsia, oriental sore, verruga peruana, tinea imbricata, dhobie itch, and uncinarial dermatitis, which have been entirely rewritten or materially altered. As a text-book and work of reference the volume is probably unsurpassed, and it will doubtless continue to enjoy the favor of the profession. A. K.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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The Relation of the Parathyroids to Calcium Metabolism and the Nature of Tetany.—W. G. MacCallum and C. Voegtlin (*Johns Hopkins Hospital Bulletin*, 1908, xix, 91) note that clinical studies of such conditions as rickets, osteomalacia, etc., have suggested the possibility that tetany might stand in some relation to disturbances of the calcium metabolism; also that calcium counteracts the effect of other salts which produce muscular twitching, and moreover that in animals, after parathyroidectomy, which are kept on an abundant milk diet rich in calcium, the tetany is either very mild or absent. This led MacCallum and Voegtlin to study the calcium metabolism of animals in which tetany is produced by parathyroidectomy, and the relations of various salts, especially those of calcium to the tetany thus produced. They conclude that all the violent symptoms caused by parathyroidectomy may be almost instantly cured by the intravenous injection of a solution of a calcium salt (either 5 per cent. of the acetate or lactate of calcium). Subcutaneous injection or taking the salt by the mouth are as effective but act more slowly. This beneficial action lasts for about twenty-four hours, when tetany may reappear; it disappears however, with the repetition of calcium therapy. Potassium salts have an opposite effect and aggravate the symptoms of tetany. These accentuated symptoms may be relieved by the calcium salts, but larger doses are required in order to overcome the added potassium effects. Magnesium has a similarly beneficial effect, but its toxic anesthetic action obscures the results.

This article is a preliminary note, and more work is being carried on along these lines by MacCallum and Voegtlin, which will appear later. That so far done indicates, however, that there is a deficiency in the calcium

content of the blood of dogs during tetany, and the excreta show an increased calcium output. Thus, apparently, the parathyroids control in some way the calcium metabolism, so that upon their removal a rapid excretion, possibly associated with inadequate absorption and assimilation, deprives the tissues of calcium salts. Thus, the administration of these calcium salts may have some therapeutic importance, not only in postoperative tetany, but also in those forms occurring spontaneously, as in children and in some infectious diseases or in pregnancy and lactation. "If further study confirms the accuracy of these observations it may be possible to compare the condition with that in pancreatic diabetes, in which the loss of control of the carbohydrate metabolism by the destruction of the Islands of Langerhans allows an abnormal carbohydrate excretion, and tetany might accordingly in that case be spoken of as a *diabetes calcareus*."

Masked Pneumothorax. C. SABOURIN (*Rev. de méd.*, 1908, xxviii, 89) gives this name to those cases of pneumothorax in which, although the symptoms and the condition of the patient point to the trouble, yet physical signs are either absent or in abeyance for a while. This may also be the case in certain central pneumonias and interlobar pleurisies, and in some instances of tuberculous cavities in which, at times, the usual physical signs are wanting. These changes from the usual pictures are brought out by the depth of the lesion. In pneumothorax, if the opening be into the mediastinum, interlobar, or toward the diaphragm, and particularly in those cases in which the area becomes more or less encysted, the difficulties of diagnosis are much greater than ordinarily. Sabourin reports four cases of tuberculosis that presented suddenly the symptoms of pneumothorax, pleuritic pain, dyspnœa, and signs of compression, displacement of the mediastinal organs and diaphragm without any auscultatory or percutatory phenomena. These signs were in abeyance for from three to six days. A fifth case of the same character is described in which there were no signs of pneumothorax before death, yet at the postmortem they were evident. In these cases the many pleuritic adhesions helped undoubtedly to mask the condition.

Heart Disease and Pregnancy.—L. POULIOT (*Arch. gén. de méd.*, 1908, lxxxviii, 65). Since 1869, when Professor Peter, after seeing a sudden death in pregnancy due to heart disease, enunciated the following so-called law, that in such conditions "girls should not marry, wives should not have children, and mothers should not nurse their children," there has been more or less discussion of the subject, some advocating these stringent measures, others more lenient except in the presence of grave cardiac lesions. These views Pouliot has carefully considered, and after a study of the subject does not think that all forms of cardiac disease forbid pregnancy. Mitral stenosis is the most serious condition, especially as acute pulmonary complications are apt to set in, œdema being the most serious. The myocardial conditions, when at all advanced, are also very serious, especially when there is marked asystole, the mortality in these cases being very high—46 per cent. in 555 cases. As regards treatment in these conditions the rapidity

of the symptoms leading up to pulmonary œdema is often so great that venesection is the best treatment. This should be followed later by cardiac stimulants and the interruption of pregnancy or the ending of labor as soon as possible. Pouliot notes, however, the great frequency of perfectly normal pregnancies and deliveries of women suffering from the various heart diseases; this is especially brought out in routine hospital examinations in which the cardiac lesions are discovered without specially suggestive symptoms. Pouliot, therefore, concludes that with the exception of the graver conditions noted above, ordinary cardiac lesions need not be a bar to pregnancy and successful labor.

Hemophilia.—Marcel Labbe (*Rev. de méd.*, 1908, xxviii, 103) considers the pathogenesis and treatment of this interesting condition. As regards its etiology, he groups the condition under four headings and summarizes briefly the different views in literature as to whether its cause lies in the heart or in the circulatory system, whether it is of nervous origin or due to some inherent trouble in the blood. This latter view he discusses most thoroughly. Of especial interest, however, is the treatment of the disease. Labbé goes over various drugs and methods and comments upon their value. The most recent and successful method of treatment is the injection of fresh normal blood as advocated by P. E. Weil, either from the horse, rabbit, or human blood. Beef serum must not be used, as it is apt to set up a febrile reaction. In case of necessity a horse's antidiphtheritic serum may be used. The dose, if given intravenously, is 10 to 20 c.c.; if given subcutaneously, this should be doubled. Serum must be fresh; that is, not older than fifteen days. After injection the coagulability of the blood is greatly increased, and this effect lasts for some weeks, although it is at its height within the first two to three days. The good effect of the serum is more evident in those cases in which the cause lies in the blood plasma rather than in the permeability of the vessel wall. In other cases the condition is benefited, but not as much. The injection may be repeated when necessary, with the same effects as in a primary inoculation. By means, then, of this serum which may be injected before surgical operations, work of this character may be done on hemophiliacs without an alarming hemorrhage. It never could be done before. Gauze soaked with the serum is of great use in minor surgical work, and has proved successful in dental surgery and in severe epistaxis.

Experimental Studies on Posthemorrhagic Anemias and Their Relation to Aplastic Anemia.—Blumenthal and Morawitz (*Deut. Arch. f. klin. Med.*, 1907, xcii, 25) first review the cases of aplastic anemia reported in the literature, emphasizing the salient clinical and pathological anatomical features of the disease. As hemorrhage has been a prominent symptom in the history of several cases, they undertook to reproduce in the lower animals by repeated bleedings, histological changes analogous to those occurring in the aplastic anemia of man. Their experiments were carried out upon dogs and rabbits. The dogs were bled daily from a vein, over a considerable period of time; with the rabbits leeches were used to remove the blood. Young animals were found to be suitable for the work because of the rapidity with which their blood is

regenerated after hemorrhage. In older animals, in which the hematopoiesis is less active, the bone marrow showed only slight hyperplasia; this was of the myeloblastic type—the predominating cell being mononuclear and non-granular, nucleated red blood cells; granulocytes were very few in number. In the spleen megalokaryocytes were found, but they saw no evidences of blood formation here. In one instance the spleen contained numerous phagocytes filled with red cells. No signs of blood formation were observed in the liver. The peripheral blood, which was frequently examined, resembled that seen in aplastic anemia in man in the absence of poikilocytosis and the small number of nucleated red blood cells, and differed from it in the presence of basophilic granules and polychromatophilia in the red cells, and in the absence of leukopenia and lymphocytosis. The basophilic granules of the erythrocytes Blumenthal and Morawitz look upon as nuclear in origin; the granules were found in the blood of those animals in which regeneration was most active; they were not found in association with aplastic bone marrow, evidence of their nuclear source.

The Diagnosis of Chronic Pentosuria.—Vas (*Wien. klin. Woch.*, 1908, xxi, 313) reviews the cases of chronic pentosuria and adds two cases of his own. The condition is a perversion of metabolism without characteristic symptoms and is not incompatible with good health. Alimentary pentosuria, which may follow the ingestion of cherries, plums, etc., bears the same relation to chronic pentosuria that alimentary glycosuria bears to diabetes mellitus, and the two conditions must, of course, be sharply differentiated. With the usual orcin and phloroglucin tests employed for the detection of pentoses in the urine, apparently positive results may be obtained from the presence of paired glycuronic acid compounds. Vas has found Bial's modification of the orcin test of value in avoiding this difficulty. (Bial's reagent consists of hydrochloric acid, concentrated 500 c.c., in which is dissolved 1 gm. of orcin; to this solution 20 to 80 drops of 10 per cent. ferric chloride are added). The rarity of chronic pentosuria is shown by the fact that Vas met with but two instances in about two thousand specimens of urine examined for pentoses.

Alimentary Lipemia.—Neisser and Brauning (*Ztschr. f. exp. Path. u. Ther.*, 1907, iv, 747) have made a very extensive study of digestive lipemia. A few drops of chyle suffice to render cloudy 100 c.c. of clear blood serum. After each meal rich in fat a fairly strong stream of emulsified fat flows into the blood from the thoracic duct, and the turbidity of the serum which is produced thereby is very readily detected by the unaided eye.

The serum of 96 patients was examined after twelve hours fasting (6 P.M. to 6 A.M.). Of these, 84 had perfectly clear blood serum, while in 11 they found a slight turbidity and in one a marked opacity. The 12 patients with turbid serum suffered from the following diseases: Diabetes, 1; intermittent glycosuria, 2; alcoholism, 4; acute nephritis, 1; adiposity, 1; carcinoma of the liver, 1; 2 had no disease. The very cloudy serum was obtained from a patient suffering from arteriosclerosis and alcoholism. Experimentally they found clear serum in six cats which had been starved forty-eight hours. They therefore conclude

that in man, twelve hours after the last meal, and in fasting cats, the serum is clear except in certain diseases. If a man has eaten a few hours before the blood is taken, the serum is nearly always cloudy. Neisser and Bräuning find, by giving specially chosen meals, that the cloudy serum is obtained only after the patient has eaten food containing fats. They now examined the serum of seventy-one patients following the administration of 100 gm. cream to which 70 gm. fluid butter had been added; turbid serum was met with sixty-seven times, clear serum four times. The patients with clear serum suffered from cirrhosis of the liver (fatty stool), cancer of the liver (fatty stool), cancer of the pancreas (fatty stool), and cancer of the œsophagus (shortly before death) respectively. In cats they always found a cloudy serum after a fat meal. Thus, they conclude that the blood serum is always cloudy a few hours after the giving of a meal rich in fat, except when there is interference with the absorption of fats. The microscopic appearance of the blood with turbid serum was striking. Fat droplets of various sizes are seen. The smallest are identical in appearance with hemokonia, and, like the latter, they show well-marked Brownian movement. Using the ultramicroscope, one can see still smaller droplets. If, now, one shakes out a turbid serum obtained after a fat meal with some ether, the serum becomes clear and the hemokonia are no longer to be seen. Allowing the ethereal extract to evaporate, a fatty residue results. Further, if 100 gm. of butter is melted and stained with Sudan III and then given to a cat, the serum taken in the next few hours will show numerous hemokonia stained red. The identity of hemokonia, is, therefore, established. By administering to the patient a fat which does not remain fluid at moderately low temperatures, the degree of the alimentary lipemia may be measured directly. The method is as follows: The patient fasts during the night. At 7 A.M. he is given 100 gm. cream and 30 gm. butter. During the remaineder of the day fat-free food must be given. Blood is withdrawn at intervals of a few hours for twelve hours after the test meal. The serum after complete separation is drawn into long, narrow, glass tubes and placed in the refrigerator. In the course of twenty-four to forty-eight hours a "cream" will collect on the surface. The height of this layer, compared to the column of serum, may be used to express the degree of lipemia.

Cellular Changes in the Blood-forming Organs.—MORAWITZ and REHN (*Deut. Arch. f. klin. Med.*, 1907, xcii, 109) have studied the myeloblastic bone marrow obtained by Blumenthal and Morawitz as a result of their work upon aplastic anemia. They distinguish the non-granular cells of the bone marrow (myeloblasts) from the lymphocytes and lymphoblasts solely by the granules in the protoplasm as shown by Altmann's stain. In smears and sections of the spleen and lymph glands one finds a few granules placed about the nucleus in the lymphocytes and lymphoblasts; in the lymphoid cells of the bone marrow (myeloblasts) these granules are completely lacking. They find the characteristics of myeloblasts as given by Naegeli, especially the number of the nucleoli, to be unreliable. Morawitz and Rehn consider the myeloblast the antecedent of the myelocyte. They found nothing to support the view that the myeloblast may represent a degenerated myelocyte.

SURGERY.

UNDER THE CHARGE OF

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Some New Facts Concerning the Physiology of the Testicles and the Prostate.—SERRALACH and PARES (*Annal. d. mal. d. org. gén.-urin.*, 1908, I, 161), in studying the syndrome characterizing prostatic hypertrophy, were impressed particularly by the effect upon the function of the bladder. In the first period, that of congestion, the contractility of the bladder is equal to and sometimes greater than in the normal condition, although in the period of chronic retention the power of the bladder is much reduced. In nearly all cases the coefficient of contractility is increased after prostatectomy. This contractility has been observed at the end of the operation during irrigation of the bladder. A similar result has been observed after castration. A patient of Albarran with complete retention for six weeks, began to urinate spontaneously five hours after double castration. The retention in this case was rather the result of an inhibitory influence than of obstruction by the prostate. The authors hold that castration may produce atrophy of the normal prostate, but not of a pathological prostate. When atrophy does occur it takes three to six months to develop, so that it does not explain the rapid return of function already referred to. This rapidity can be explained best by a nervous effect. The effect produced by double castration is more marked than that by double vasectomy. The authors refer to two cases in which acute retention of urine had developed without apparent cause. In each case there was a history of sexual excess on the night before the development of the retention. They believe that an inhibitory effect is produced on the bladder by the testicles, and that this effect is removed by castration. From their experimental researches they are convinced that the prostate has an internal secretion which so acts upon spermatogenesis, and even upon the life of the testicle, that when one removes the prostate, atrophy of the testicles soon develops. This effect is indirectly like that of castration. The essential incontinence of children disappears spontaneously somewhere between one and fourteen years of age. The latter is the age at which the testicles usually begin to functionate, and thus to exert their inhibiting influence on the bladder. As the result of their experiments Serralach and Parés concluded that the testicles secreted a substance, X, which, when transferred into the circulation influences the contractility of the bladder. Its effect is to contract the membranous

urethra with more force than the neck of the bladder. It provokes relaxation of the bladder muscle. It increases the capacity of the bladder and delays the necessity for urination.

The Treatment of Spondylitis.—Wiener (*Zentralbl. f. Chir.* 1908, xxxv, 252) says it is generally recognized that complete immobilization of the spine impairs seriously the respiration and digestion. The prevention of muscle spasm is sufficient. Wiener speaks favorably of Hessing's corset, and says that with it firm kyphosis may apparently be diminished by the addition of elastic traction. The cases in which the first four dorsal vertebræ are involved give the worst prognosis. Wiener reports a case of this kind in which a new method of treatment proved satisfactory. A woman, aged fifty-four years, had suffered for fifteen years from this condition. For a year she had experienced pain in the back which was transferred to the head, shoulders, and abdomen, and for six months she had been unable to get out of bed. The lower extremities could not be moved actively, and there was "foot drop," œdema of the feet and lower part of the legs. Clonic spasms of the legs occurred every few minutes, and these were accompanied by severe pain requiring heavy doses of morphine. These were due to the associated and renewed pachymeningitis. The pain was immediately relieved by weight extension to the head. At night a ten pound weight extension was applied in the usual way. During the greater part of the day the patient was placed in an easy chair and the weight extension was effected through a pulley in the ceiling. The patient was now able to perform active movements. To protect the head from being struck by the weight a special apparatus was suspended from a steel wire stretched tightly between two rooms. According to the needs of the patient the weight could be increased or decreased quickly by bags of shot weighing from one to five pounds. Wiener did not find it necessary to exceed ten pounds for the extension. The patient by means of this apparatus was moved from one part of the room to another. In a few days the clonic spasms were more rare and the first active movements occurred. After four weeks the patient could pass rapidly from one room to the other with the ten pound weight extension applied. In four months she could move freely, and in seven months she was discharged as completely healed.

The Origin of Median Cervical Fistulæ.—Weglowski (*Zentralbl. f. Chir.*, 1908, xxxv, 289) calls attention to the fact that His, in 1880, showed that the origin of median cervical fistulæ was associated with the development of the median portion of the thyroid gland. It has not been explained why sometimes these fistulæ are lined with a single layer of epithelium, and at other times with several layers, and why its walls contain mucous glands, lymphoid follicles, etc. After a series of investigations upon 36 human embryos, 92 children, and 25 adult cadavers, Weglowski reached the following conclusions: In men, from an incomplete development of the median portion of the thyroid gland (in more than 30 per cent. of the bodies examined), there was found the remains of the primitive passage from the gland to the root of the tongue. This was either in the form of a separated small portion of the gland, a canal, or a cyst. These remains are usually not perceptible,

but may under favorable circumstances lead to the formation of goitres in unusual situations, or to fistulæ and cysts, the latter often becoming fistulæ later. The complicated microscopic structure of the fistulæ (single and several layered epithelial lining, lymphoid follicles, etc.) takes its origin from the mucous membrane of the root of the tongue, the isolated elements of which are drawn downward into the tissues by the developing median portion of the thyroid gland.

A Communication on a Case of Operative Cure of Venous Thrombosis in the Region of the Superior Mesenteric Vein.—HAAGN (*Deut. Zeit. f. Chir.*, 1908, xcii, 79) says that this is a very rare condition, and that the prognosis is very bad whether treated medically or surgically. The diagnosis in Haagn's case was made at the time of operation. The patient was a woman who had borne five children, the last being one and one-quarter years of age. After this birth she had suffered severe child-bed fever. The attending physician diagnosticated pelvic cellulitis. The patient was ill for a half year. On the day of admission to the hospital, at 7 A.M., she was taken with sudden and severe pain in the abdomen, repeated vomiting, and no passage of flatus. A purgative produced two pulpy stools. Morphine did not control the pain. Examination revealed a specially painful area on the left side. During the manipulations the patient experienced a sudden movement, when the pain disappeared. The patient looked very ill, temperature 37.5°, and pulse 80. There was only slight distention, and to the left of the umbilicus could be palpated a sausage-shaped resistance, tender to the touch. Following a desire to defecate, she passed about a litre of pure fluid blood. Operation was delayed because of the improvement. The next day the symptoms were worse, and laparotomy was performed on the succeeding day. A median supra-umbilical incision permitted the escape of distended intestines, from among which there escaped a brownish red fluid. A sausage-shaped swelling which proved to be part of the jejunum, 20 to 30 cm. long, bluish red in color, without motion, and its walls several times their normal thickness was found on the left side. The corresponding portion of the mesentery showed numerous thrombi. The affected area formed a triangle, the base of which was at the intestine, the apex at the root of the mesentery. The peritoneal surface was lustreless and abundantly covered with fibrinous deposits. The ascending colon was drawn toward the attachment of the affected portion of the mesentery by a band, which was ligated and divided. The affected mesentery and bowel were resected and the divided ends united by a Murphy button. Drainage was employed. Eleven days later the presence of an intestinal fistula was recognized. Twenty days afterward the abdomen was re-opened on account of the symptoms of ileus. Several coils of intestine were found adherent to one another and one portion of bowel seemed to be acutely kinked, the proximal portion being distended. While correcting this flexion by separating adhesions, an opening was torn in the bowel wall. This was closed and the proximal portion of the bowel sutured into the abdominal wound for the performance of an enterostomy. Owing to the weakness of the patient the intestinal fistula was not closed. Seven months after the operation the patient was completely cured and could perform her usual work.

The sudden onset with vomiting and complete bowel obstruction made one think of strangulation ileus. The improvement in a few hours and the bowel movement, however, excluded it. Intussusception may develop in an elderly person, and, as in this condition, the bowels may move and blood be passed. This was Haagn's diagnosis. Kussmaul gives the symptoms of thrombosis of the mesenteric vessels as follows: Evidence of the source of the embolus; abundant hemorrhage from the bowel; subnormal temperature; peritoneal symptoms, as vomiting and abdominal pain. Haagn says that operation should not be done when the patient is in severe shock or has general peritonitis, and not as long as symptoms of paralytic ileus continue. Above all, one should determine as far as possible the probable extent of the gangrenous tissue. The best indication for immediate operation is the beginning of peritoneal symptoms. When anastomosis of the ends of the bowel after resection is possible, and one is sure that he has gone well into healthy bowel, this operation is to be preferred to a preternatural anus.

Syphilitic Dactylitis.—CREITE (*Deut. Zeit. f. Chir.*, 1908, xcii, 70) believes that this affection begins in the soft tissues of the fingers, that it extends to the finger joints and bones, and finally results in considerable defects in the diseased bones. There develops slowly, with boring pain, a swelling in one finger joint, usually in the middle one. It is of a solid, almost bony consistency, is slightly bluish in color, and covered by tense, smooth skin. With variations in the degree of swelling there develops, in the course of weeks or months, an acute condition with more marked swelling, severe pain, and bluish discoloration of the skin. There is now seen a small vesicle about the size of a millet seed, with clear, watery contents, which, with the abatement of the acute symptoms dries up and in a few days disappears entirely. There is no perforation of the skin with ulceration and fistula formation. Rather does the swelling become smaller and the neighboring joints diseased and stiffened. The disease extends to the bones, with the development of defects in them and of shortening and deformities of the fingers. There was little or no effusion in the joint in Creite's case, but the surrounding soft tissues were actively involved in the inflammatory process. The joint did not suppurate, but the cartilages were involved and were quickly and completely absorbed. The original swelling of the bone was the result of a gummatous formation, which so affected the bloodvessels as to disturb the nourishment and lead to absorption of the bone without suppuration. As this rarifying osteitis proceeds, the inflammatory symptoms subside and disappear entirely, the color gradually becomes normal, and from the absorption of the bone the finger becomes considerably shortened.

A Case of Primary Renal Actinomycosis.—KUNITH (*Deut. Zeit. f. Chir.*, 1908, xcii, 181) says that while actinomycosis of the kidney, secondary to some other focus, has been reported several times, up to the present only one case (von Israel) of the primary renal actinomycosis has been published. Kunith now reports a second case which occurred in a boy, aged four and three-quarters years, who, like v. Israel's patient, was operated on with successful result. The parents were healthy, but the grandparents and an uncle on the mother's side had died of phthisis. The

boy had had the ordinary diseases of childhood, and had always lived in Berlin, except for a stay of five or six weeks in the country when he was one and one-half years of age. In April, 1906, he fell from a swing, and as a consequence developed a swelling in the lumbar region. This swelling was twice incised by a physician, with the escape of pus. Two fistulæ resulted which would not heal. Four months before admission the patient suffered from temporary bladder trouble, and more recently the urine has been turbid, blood being passed on one occasion. For this he was brought to the hospital. He was slender, poorly nourished, and pale. His temperature and pulse were normal. The molar teeth of the lower jaw were carious, but the jaw was not swollen or carious. The heart and lungs were apparently normal and the abdomen soft. In the right kidney region was felt a firm resistance, and in the back on the same side below the tip of the twelfth rib was a granulating, suppurating fistula, and close by a second one. Below was a scar. The secretion from the fistulæ was very scanty, seropurulent, but without other peculiarities. A scab soon formed, and when a probe was introdueed it passed only a short distancc. A probable tubercular kidney was diagnosticated and this was removed by what proved to be a rather difficult operation. In about four months the patient left the hospital completely healed, and eleven months after admission he had no further trouble and had improved very much in weight. From the fourth day after operation, for about five weeks, ray fungi were found in the urine. These consisted of an entanglement of threads, usually surrounded by a group of leukocytes. Clubs were found only twice, and these soon after the operation. Besides the actinomyces, the urinary sediment contained an abundance of leukocytes, blood for a short time after operation, and for a longer time hyaline and granular casts. Albumen disappeared completely from the urine only after four months.

The Permanent Results in Tuberculous Coxitis.—PERRET (*Archiv f. klin. Chir.*, 1908, lxxxv, 561) says that tuberculous disease of the hip-joint has usually a very gradual, rarely a violent, acute origin. Direct and indirect traumatism, most frequently of mild grades, are the usual causes; infectious diseases and metastases more rarely. There is throughout, especially in young individuals, a considerable disposition to spontaneous cure; yet the frequency of relapses is a warning against a favorable prognosis. In young individuals the infection of the joint is secondary to a primary osteal focus; while in advanced age it is of synovial origin, except in those cases in which an old encapsulated, latent, osteal focus after years again becomes active. Tuberculous foci not in the wall of the acetabulum, but in its immediate vicinity, may invade the hip-joint; usually they do not. Primary foci in the head or neck of the femur sooner or later, as a rule, break into the joint. A coxitis of pelvic origin may have in the early stages, apparently, as favorable a prognosis as that of femoral origin. In the later stages, however, it develops an extensive caries of the acetabulum, in which resection of the joint may be without good results. The best functional results were obtained by conservative operations, especially by early arthrotomy with sequestrotomy or erasion of the tuberculous focus. Conservative treatment gave good results, although it was not always sufficient, and

in half of the cases it was necessary to substitute conservative operations, especially resection. In mild, favorable cases with or without enclosed suppuration, in which the seat of the focus cannot be determined with certainty either by clinical examination or by the *x*-rays, the treatment should be conservative until the osteal focus is evidently not undergoing spontaneous healing, but is recognizably extending. In neglected severe cases, with open sinuses and with other threatening complications, arthrotomy for the exploration of the joint should be carried out. When the joint is so destroyed that there is no hope of spontaneous healing, all the diseased tissue should be thoroughly removed and the healthy tissue saved, "atypical resection." Perret hopes that typical resection, that is, decapitation or still more, subtrochanteric excision, will gradually disappear. When by an exact clinical investigation the tuberculous focus can be localized, and this can be confirmed by the *x*-rays, our present day aseptic arthrotomy with sequestrotomy, or erasion of the diseased part, will give the best results from the standpoints of time, radical healing, and functional results.

THERAPEUTICS.

UNDER THE CHARGE OF

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Oil Injections in Constipation.—VIDAL (*Journal de médecine de Paris* (1908, xx, 2) advises the use of irrigations of sterile alcohol-free oil in constipation. A fountain syringe is employed to which a soft rubber catheter, which is passed about 3 inches into the rectum, is fitted. The oil is introduced about two hours after the evening meal, the patient lying on his back or left side. The quantity given is from about 2 to 7 ounces, and its temperature should be about 95° F. After the tube is inserted the reservoir of the syringe is elevated to about 3 feet above the patient and the oil is allowed to flow slowly into the rectum. Should there be difficulty in inserting the tube, the rectum should be cleared by means of an irrigation of saline solution. The oil should be retained all night, and in the morning, on rising, an irrigation of about a pint of saline solution at 100.5° F. is taken. This should be retained for a few minutes. The oil injections are given daily at first, but after a week the intervals may be lengthened until one every five days becomes sufficient. The treatment may, at first, cause colicky pains, but these soon cease.

Flexner's Serum in Epidemic Meningitis.—Dunn (*Boston Med. and Surg. Jour.*, 1908, clviii, 370) has used Flexner's antiserum in 15 instances of epidemic cerebrospinal meninigitis, in all but one of which the diagnosis was confirmed by finding Diplococcus intracellularis in the cerebrospinal fluid. Of these instances, 8 have resulted in complete recovery, 2 have died, and 5 are still pending. The 8 patients who have recovered are all perfectly well, having been left with no sequels of any kind. The 2 fatalities were both in chronic infections, in which the disease had run considerable time before coming under observation. Of the 5 instances still pending, 4 will undoubtedly recover; the other is a chronic infection, and in it the outcome is dubious. Each of the 8 patients who received the serum in the first week of the disease has completely recovered, and of the pending instances, 2, in which the antiserum was given early, are convalescent. The most important point suggested by the results attained is the advantage to be gained by giving the serum early in the disease, and it seems impossible to overestimate the value of early diagnosis and treatment. It would seem that the results obtained with this serum in the patients reported are sufficiently good to afford strong basis of hope that this treatment will prove of a value commensurate with that of the antitoxin treatment of diphtheria, and, although the treatment requires further testing, the author believes that the serum should be used in every instance of this disease and as early as possible. The serum is injected into the spinal canal by means of a lumbar puncture. As much fluid as will run freely is allowed to escape, then the syringe is filled with the antiserum and is connected with the needle through which the fluid has escaped, and the serum is injected. Flexner suggests 7½ drams as the maximum dose, to be repeated for three or four days, and that at least as much cerebrospinal fluid as this should be first withdrawn in order to avoid risk of unduly increasing the cerebral pressure.

The Toxicity of Therapeutic Sera.—Besredka (*Revue médico-sociale*, 1908, ii, 10) holds that the toxicity of therapeutic sera should be tested by making injections of the sera into the brains of guinea-pigs. Such tests show that different sera possess different powers of toxicity, the fatal dosage varying from $\frac{1}{4}$ c.c. to $\frac{1}{128}$ c.c. Sera from horses living under similar conditions possess practically the same toxic powers, variations being rare and of little importance. Variations in the toxicity of sera seem to be due, first, to their origin, second, to their age. A serum hypertoxic when fresh loses, little by little, its toxic qualities; this loss, rapid at first, gradually becomes slower, and ceases by the tenth day. All therapeutic sera should, however, be considered to possess toxic qualities for two months after they have been drawn. In general, every serum which causes grave anaphylactic phenomena in doses of $\frac{1}{10}$ to $\frac{1}{20}$ c.c. or less should be considered as toxic. The technique of the intracerebral injection is simple, rapid, and not expensive.

Bacterial Vaccines in Septicemic Conditions.—Bristow (*New York State Jour. Med.*, 1908, viii, 120) reports the results in four patients in whom vaccine treatment was attended with excellent effects. Two of the patients suffered from a streptococcic septicemia, a third was afflicted with furunculosis, and the fourth possessed a gonorrhœal arthritis.

Each of these was treated with injections of an appropriate vaccine. No conclusions are drawn, since the number of patients is so small, but the fact that the vaccine treatment succeeded when other measures had signally failed is most suggestive.

Colloid Metals in Therapeutics.—CAPEZZUOLI (*Zentralbl. f. die ges. Therapie*, 1908, xxvi, 113), in a paper under this title, cites 24 instances of appendicitis, reported by Moosbrugger, which were treated by colloidal silver. Of the 24 patients, only 2 died, and these exhibited the symptoms of severe peritonitis before treatment was begun. The colloidal silver was given internally in doses of a tablespoonful every half to one hour of a 0.5 to 1.5 per cent. solution; twice a day 30 grains of unguentum Credé was rubbed into the skin, and twice daily enemas of 7½ grains of colloidal silver in 3 ounces of water were given. Children received one-fourth of the above doses. In most of the patients improvement was evident in two days, and upon the third the fever and local symptoms abated. In the severer instances improvement was evident in from four to eight days. The author compares the action of colloidal silver in appendicitis to that of antitoxin in diphtheria.

Intravenous Injections of Colloidal Silver.—FRANCKE (*Medizinische Klinik*, 1908, iv, 12) states that in grave infectious processes, such as sepsis, puerperal and otherwise, colloidal silver should be employed in 4 to 5 per cent. solution as an intravenous injection, 30 grains being injected daily or every other day, according to the type of the affection and the effect of the remedy. In one instance of severe gonorrhœal sepsis, with chills persisting for weeks, one such injection brought about a rapid and permanent fall of temperature. Injections of colloidal silver are usually followed by a chill succeeded by a rise of temperature even as high as 105.8° F., but this phenomenon has no untoward sequels. The employment of this agent by intravenous injection is superior to its exhibition by enema or inunction, and the former is the preferable mode of administration.

Quinine Hydrobromide in Exophthalmic Goitre.—JACKSON and MEAD (*Boston Med. and Surg. Jour.*, 1908, clviii, 346) recapitulate their results with this drug as follows: 42 patients cured (no signs or symptoms for two years), or 76 per cent.; 7 patients benefited, or 13 per cent.; 6 failures, or 11 per cent. It is not claimed that the cures are permanent, yet the patients are free from symptoms and signs and are able to perform their work. In addition to the use of the quinine, general treatment, such as rest, diet, care of the stomach, intestines, and skin, was instituted. Quinine hydrobromide is a combination of quinine and hydrobromic acid in which all the bonds are satisfied and we have a neutral salt with a formula, $C_{20}H_{24}N_2O_2.Br + H_2O$. In order to get the best results the salt must be the neutral, not the acid salt. The dosage is 5 grains in capsules three times a day; this is as much as is ordinarily well borne, but some patients may be able to take 4 capsules daily without tinnitus; others cannot take more than 2 capsules a day. The patient should be told that he should expect little or no benefit short of a month, and that the treatment must be continued for at least two years. Usually, after a week or two the pulse rate will be slowed,

the thyroid diminished, and the sweating or tremor lessened. The treatment should be continued until all the symptoms have disappeared, which may be in four months or not for three years. No bad effects have been noticed to follow the treatment, and the only unpleasant action of the drug is occasional tinnitus, especially if large doses be given. Relapses are not rare, but if taken in hand quickly usually yield readily. The author does not attempt to explain how the quinine acts, but is convinced that it has a distinct curative effect.

The Physiological Action of Iodothyrin.—Von Furth and Schwartz (*Klin.-therap. Woch.*, 1908, xv, 209) have examined animals in which a condition of hyperthyroidism had been induced by means of the administration of thyroid preparations (iodothyrin) and in which Loewi's phenomenon (pupillary dilatation following instillation of adrenalin into the eye) had been induced. The results of the experimentation appeared to show that iodothyrin is not the only principle contained in the thyroid gland. After intravenous injection of iodothyrin in cats the blood pressure falls and the pulse rate becomes less, due to stimulation of the vagus centre in the medulla; even temporary cessation of the heart action may be induced. Artificial iodothyrin has the same action; iodothyrin is a complex iodine derivative which is contained in an albumin molecule.

Drugs in Diabetes.—Elliott (*Illinois Med. Jour.*, 1908, xiii, 312) considers that it is inviting failure to give a drug cure to a diabetic, and by beginning the treatment with drugs, the patient, ever prone to believe in the occult power of medicine, comes by degrees to rely upon this part of his treatment rather than upon the infinitely more valuable dietary rules; he naturally tends to follow the path of least resistance, and prefers his capsules or drops to the more arduous self-denying system of diet. Of the drugs usually given, opium is the first on the list, but its long-continued and indiscriminate administration is unjustifiable. It exerts no specific action, and its effects on a given patient cannot be foretold. It may predispose to coma, and there is the danger of habit formation. Antipyrine, acetanilide, aspirin, and the salicylates may depress the patient, cause cardiac irritability, and have been known to induce albuminuria. In gouty patients the salicylates may be of benefit. Jambul has never proved of use in the author's hands. Arsenic has no specific effect, but small doses act well on general nutrition, and may do good. The alkalies are of distinct value. They may be given in the form of mineral waters or as one of the medicinal salts, carbonates, bicarbonates, citrates, or tartrates, the sodium salts being preferable. The dose will depend on the stage of the disease and the degree of the acid toxemia. In severe types of the latter alkalies act as a prophylactic of coma, and must be given in large doses. Patients whose urine contains acetone should receive at least a half-ounce of sodium bicarbonate daily, and if coma is impending two to four times this amount should be given. In constipation sodium citrate may be added to the bicarbonate with advantage, and the additional employment of calcium carbonate to replace the calcium waste characteristic of diabetes has been suggested.

PEDIATRICS.

UNDER THE CHARGE OF

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Needle in the Left Bronchus.—H. VON SCHROETTER (*Deut. med. Woch.*, 1908, xxxiv, 641) reports the case of a girl, aged twelve years, who had swallowed a shawl pin, 74 mm. (3 inches), long, the glass head of the pin had a diameter of 9 mm. The pin entered the left bronchus head first, the latter being in one of the divisions going to the lower lobe at the time the Röntgen picture was taken. Twenty-one days after the accident Schroetter removed the pin by bronchoscopy using a tube 21 cm. long and 8.5 mm. in diameter. Local anesthesia was employed and the needle grasped at the third attempt and pulled out without injury to the bronchial structures. No disturbances developed, the patient remaining perfectly well.

The Treatment of Diphtheria with Pyocyanase and the Persistance of the Diphtheria Bacillus.—P. L. SCHLIPPE (*Deut. med. Woch.*, 1908, xxxi, 588) has employed pyocyanase in the treatment of 54 cases of diphtheria of varying degree. Several cubic centimeters of pyocyanase were dusted energetically upon the diseased portion of mucous membrane four times daily, the treatment being divided into two parts, with an interval of five to ten minutes between applications. According to various observers the favorable action of pyocyanase depends on its bactericidal action, in its power of impeding the development of bacteria, of neutralizing their toxins, dissolving the diphtheritic membrane, destroying the pathogenic organisms upon which the mixed infection depends, and, finally, upon favoring the restitution of the mucous membrane. In a number of cases no antitoxin was employed; in some of these, and particularly the lighter cases, the membrane disappeared very quickly and the symptoms diminished with it; in one instance only one side was thus treated and the membrane persisted on the untreated side. A piece of membrane treated with pyocyanase in a test-tube at blood heat dissolved in a similar manner. In other cases, however, the pyocyanase did not seem to suffice and antitoxin had to be injected, as the process spread further, or the toxic symptoms increased to an alarming degree. All these patients improved at once as soon as the serum was injected; 24 of the patients were treated with serum and pyocyanase simultaneously. In 9 the specific action of the powder was noted, in 9 others it was not, and 6 died in spite of both measures. When the trachea was affected the pyocyanase blown into the trachea through a cannula did not influence the course of the process. Concerning the persistence of the organism 46 patients were studied; in 4 no bacilli could be found after the first week, in 3 after the second week, in 5 after the third, and in 5 after the fourth week; in all others the bacilli persisted more than a month and in some considerably longer,

even if pyocyanase was blown into the nose and pharynx. Chronic diphtheria developed in a number of them. This persistence is probably due to the fact that the pyocyanase cannot enter all the crypts of the nasopharynx in which the bacilli vegetate. Schlippe comes to the following conclusions: Pyocyanase must never be used in the treatment of diphtheria without simultaneous injection of serum. In some cases the pyocyanase seems to quicken the solution of the diphtheritic membrane, to remove the fœtor ex ore at once, and to produce immediate subjective improvement. It deserves to be employed further, therefore, in cases of intense membrane formation and when the membrane persists abnormally long. In very grave cases of septic diphtheria the pyocyanase does not seem to have any influence. Pyocyanase does not prevent the persistence of the diphtheria bacilli, nor does it kill the bacillus in instances of marked persistence and chronic diphtheria.

Bradycardia in Appendicitis of Children.—J. VON BOKAY (*Deut. med. Woch.*, 1908, xxxiv, 649) reports briefly the histories of 10 cases of appendicitis in children, all of whom presented marked bradycardia at some time during the disease. He deduces the following conclusions from the study of his cases: (1) During the period of absorption of peri-appendicular inflammatory exudate bradycardia may be said to be almost the rule and may exist over several days, even more than two weeks. (2) It may also appear after incision of an appendicular abscess and may persist eventually for days. (3) It may also follow resection of an appendix when the operation is performed later during the quiescent stage and may then also persist for some time. (4) The number of pulse beats may fall in children of between seven and sixteen years to 52; in most of Bokay's cases the pulse varied during the period of bradycardia between 52 and 80. (5) The pulse and temperature curves during the period of bradycardia do not run parallel to each other; hyperpyrexia was never observed when the pulse had fallen to the minimum. (6) This bradycardia connot be considered as an unfavorable symptom from a prognostic standpoint; in cases of peri-appendicular exudate it is even to be considered as a sign of a beginning absorptive process.

The Late Results of Tracheotomy.—W. WOLF (*Deut. med. Woch.*, 1908, xxxiv, 725) reports that at Trendelenburg's clinic tracheotomy was performed in 404 children between the years 1895 and 1906; 140 (31.3 per cent.) of these died, but in no instance could the fatal termination be laid to the operative interference. He compares this mortality with that of 1539 cases, in which intubation was performed and in which the fatality percentage amounted to 31.7 per cent., thus showing that the mortality from the two operations is the same. Of the remaining 264 children personal examination or letter reports were had of 173. Subsequent to the diphtheria there were 4 deaths from other causes, 145 presented no disturbances whatever, and only 24 (14.2 per cent.) had disturbances of varying degrees. In 7 the disturbance was of a more serious nature, it being tuberculosis, however, only in 4 cases, and 3 of these had an inherited disposition to the disease. Even if the disturbance in all of these 24 individuals could be traced directly to

the tracheotomy (which seems highly improbable), this figure shows conclusively that tracheotomy is not followed in many cases by late and serious indisposition. In no instance did a cicatricial stenosis develop, which, according to the opponents of tracheotomy, is a very common sequence. In the paper on intubation above mentioned, Wolf records 16 cases of cicatricial stenosis due to pressure decubitus. The splendid showing at Trendelenburg's clinic is probably due to the fact that he always performs inferior tracheotomy, which predisposes less to stenosis than superior tracheotomy. The statement made by the defenders of intubation that tracheotomy should never be performed because of the possibility of the cicatricial stenosis developing, or of a possible injury to the deeper-lying air passages (due to loss of heat which inspired air ordinarily gets in the nasopharynx) is negatived by Wolf's statistics.

The Pepsin Secretion of the Healthy and the Sick Nursling.—The method employed by J. ROSENSTEIN (*Berl. klin. Woch.*, 1908, xlv, 542) follows the principle that castor oil made turbid by the addition of hydrochloric acid is cleared up only by pepsin: filtered gastric juice was diluted in varying degrees and experiments performed to show at which dilution a certain quantity of turbid castor oil solution was still clarified. He found that the amount of pepsin in the healthy, artificially nourished infant increases with age until after the end of the third month of life; from then on it remains constant. Healthy, breast-fed infants seem to produce less pepsin, than healthy, artificially fed children of the same age. Older nurslings of less weight than normal produce the amount of pepsin which corresponds to their age. Disturbances in nutrition do not influence pepsin secretion markedly; if decomposition be present a lessening in the amount of pepsin is noted. The lack of ferment (particularly the lack of pepsin) is of slight symptomatic importance and is certainly not an etiological factor in the production of digestive disturbances of nurslings.

The Value of Pirquet's and Wolf-Calmette's Reaction in Children.—R. BING (*Berl. klin. Woch.*, 1908, xlv, 546) has employed Pirquet's test in 241 patients, 19 of whom were certainly tuberculous, 36 suspicious, and 186 not tuberculous. A positive reaction was obtained in 14 of of the first (73.7 per cent.), in 25 (69 per cent.) of the second and 34 (18.2 per cent.) of the third. Wolf-Calmette's reaction was tested in 100 cases, which were distributed over the three classes as respectively 6, 15, and 79 patients; positive reaction was obtained in respectively 3, 6, and 1 patients. He therefore considers both methods valuable and especially are the positive results in early childhood to be valued as regards the diagnosis of tuberculosis; a negative result does not necessarily mean absence of tuberculosis. If both methods show a negative result in clinically doubtful cases, the probability of tuberculosis is very likely. Pirquet's test is valuable also in cases of latent tuberculosis. Pirquet's method is without danger; the conjunctival reaction, however, is sometimes followed by violent inflammation and other unpleasant sequels, which have made Bing cautious in its use, and in the case of scrofulous children have forced him to forego it absolutely.

OBSTETRICS.

UNDER THE CHARGE OF

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The Healing of Pubiotomy Wounds.—OBERNDORFER (*Zentralbl f. Gynäk.*, Nr. 7, 1908) contributes an interesting paper with macroscopic and microscopic illustrations upon this topic. He reports the case of a multipara who was delivered of a child weighing nine pounds, by pubiotomy upon the right side of the pelvis. The patient recovered from the operation, but died fourteen months afterward from some intercurrent disease. The pelvis was secured for examination. On studying the specimen by the *x*-rays, it was found that the wound inflicted by the saw had healed without the slightest appearance of the formation of bone. Microscopic examination of the specimen showed that there was no deposit of true bony tissue. This seemed to have been prevented practically by fibers of elastic and connective tissue which extended throughout the substance between the ends of the bone. This seems an unusual result, as in the case of fractures in young individuals bony tissue is rapidly deposited, although the separation of the fragments may have been greater than after the operation of pubiotomy. The most reasonable explanation of the difference lies in the fact that the pubiotomy wound has sharply defined edges, while in the case of fracture splinters of bony tissue, small portions are irregularly projecting, and thus form centres of ossification. There seems to be no essential change in the condition of the parts as late as a year after the operation.

Chorio-epithelioma Developing after a Full-term Pregnancy.—WENCZEL (*Zentralbl. f. Gynäk.*, Nr. 7, 1908) reports the case of a woman who had borne a healthy, vigorous child at the normal period of gestation. Six weeks after labor she had irregular hemorrhage, for which a curetting was done, a large quantity of necrotic tissue, apparently connective, being removed by the curette. The irregular hemorrhage continued, and the curette removed necrotic tissue from the interior of the uterus. On examination, this proved to be chorio-epithelioma. In order to be sure of the diagnosis before sacrificing the uterus, a second curetting was done, with the same result. The uterus was then removed. Eight months afterward the patient was well. The interesting point in the case consisted in the development of the malignant growth after a perfectly normal pregnancy going to full term. The diagnosis was based entirely upon the microscopic examination of the tissue removed, as the patient presented no symptom of the condition.

Pregnancy Complicated by a Necrotic Myoma.—SCHENK (*Zentralbl. f. Gynäk.*, Nr. 7, 1908) reports the case of a patient pregnant for the first time, between the seventh and eighth month, whose pregnancy was complicated by a myomatous tumor. On examination, the abdo-

men was much distended; the tumor was composed of two portions, the lower of which extended four fingers' breadth above the pubes and seemed to contain the fœtus. The upper portion of the tumor was softer in consistence, and separated from the lower by a groove in which the finger could be laid. The condition of the patient improved very considerably, with rest in bed; the pulse and temperature became nearly normal, and operation was finally performed. The uterus was amputated at the vaginal junction and the right tube and ovary were left. The patient made a good recovery. On examination, the upper portion of the tumor was found to be a necrotic myoma which comprised four-fifths of the entire mass. In the lower portion was a fœtus about five months in development. The tumor had grown very rapidly during pregnancy, had prevented the development of the child, and rendered spontaneous labor impossible.

Retention of the Fœtal Membranes and its Treatment.—Louros (*Zent. f. Gynäk.*, Nr. 7, 1908) reports 7 cases of retention of the fœtal membranes, and concludes that several interesting questions arise in connection with this subject. He believes that just as much pains should be taken to secure the entire removal of the membranes as of the placenta. He also believes that the same care should be exercised in examination, and that should a diagnosis of retained membranes be made, the hand or sufficient fingers should be inserted, under antiseptic precautions, to remove them completely. If a very small portion of the membranes is left behind this may be allowed to remain, as it will be finally brought away in the lochial discharge. If during the pregnant period hemorrhage, fever, and foul lochia develop, the uterus should be explored to discover and remove the retained membranes. In cases in which the placenta has been expelled, but the membranes remain held within the uterus, the operator must use great caution lest the membranes be torn away from the placenta and a portion retained.

Recent Advances in Obstetrics.—Cragin (*Amer. Jour. of Obstet.*, March, 1908) contributes an article upon this subject, calling attention to recent advances in obstetrics, with a better knowledge of obstetric pathology, a better knowledge of the mechanical problem of delivery, and a better procedure. He draws attention to the pathology of toxemia, which he divides into the nephritic and hepatic types. He also recognizes pernicious nausea as a toxemic process. His paper contains illustrations of microscopic slides from the kidney and liver, illustrating the points studied. He believes that a second important advance in pathological knowledge is our increased knowledge of chorio-epithelioma malignum, especially in the matter of its diagnosis.

In the pathology of puerperal infection, we have come to appreciate the ease with which infection from without can be conveyed to the vagina or uterus, and also the danger of manipulation of the uterus in sepsis, which may change a local into a general infection.

Under the heading, "A Better Knowledge of the Mechanical Problem of Delivery," Cragin calls attention to the fact that we must not rely upon external pelvimetry, but must study the size of the fœtal head and its adaptation to the pelvic brim and cavity. Improvement in the

management of posterior rotation of the occiput through an early diagnosis and methods adapted to secure anterior rotation, are also of importance. Careful watching of the fœtal heart during labor, the cephalic application of the forceps, and in some cases rotation by forceps, are all indicated.

The importance of the nitrogenous partition of the urine is emphasized, and the fact that laboratory findings alone do not give ground for a choice of treatment unless taken in connection with the clinical study of the case. In pernicious nausea, irrigation of the colon, rectal feeding for a short time only, and early emptying of the uterus are the methods of treatment which have given the best results.

When menorrhagia or metrorrhagia persists after labor, chorio-epithelioma should be suspected, and early operation performed if a diagnosis can be made.

In the treatment of septic infection, uterine manipulation should be avoided when once the uterus has been relieved of that infected material which is ready to be discharged. The induction of labor in patients who have had difficulty in previous parturition, should be chosen when it is evident that the head is not proportionate to the pelvic canal. In cases in which the pelvis is not unusually large the prolongation of pregnancy should not be allowed to reach an extreme limit. Craniotomy has been made an infrequent operation by the improvement in Cesarean section and methods of inducing labor. Pubiotomy in hospital practice will save some children who might otherwise be lost. When mechanical dilatation is impossible, vaginal Cesarean section may be chosen.

Cragin believes that in proper surroundings, with good light and proper assistance, a well-trained man may repair with advantage laceration of the cervix immediately after labor. Unless these conditions are present, he believes that the cervix should be closed by suture in cases of hemorrhage only.

Extra-uterine Pregnancy.—Iwasse (*Archiv f. Gynäk.*, 1908, lxxxiv, Heft 2,) reports 38 cases of tubal gestation in the clinic at Tokio. His conclusions from the study of these cases are as follows: Comparing these cases with those reported in European clinics, he finds no especial difference in the origin or manifestation of ectopic gestation among Japanese and Europeans. This condition is most frequent between the twenty-fifth and thirtieth years of life; 86.8 per cent. of the patients had not previously borne children. Extra-uterine pregnancy was frequently preceded by a period of relative sterility. Its occurrence was also frequent after abortion. It seems probable that operations for correcting the position of the uterus, such as ventrofixation or intraperitoneal shortening of the round ligaments, may tend to produce ectopic gestation. Tubal abortion is most common during the first and second months of ectopic gestation, and rupture during the third and fourth months. Abortion is more frequent than rupture. When extra-uterine pregnancy goes to viability the fœtus is more apt to develop malformations and anomalous positions and presentations. Hemorrhage from the uterus and pain in the abdomen are important symptoms, usually appearing together, or separated by a short interval—not more than a week. Migration of the impregnated ovum in extra-uterine pregnancy is rarely observed.

GYNECOLOGY.

UNDER THE CHARGE OF

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Kraurosis Vulvæ.—A. W. RUSSELL (*Glasgow Med. Jour.*, December, 1907) attributes the pruritis accompanying kraurosis vulvæ to discharges from the tumor or to diabetic urine. In the treatment he recommends physiological rest of the parts, thorough cleanliness, the care of uterine, intestinal, or vaginal disorders, local application to the vulva, and excision of the parts. He advises excision. The radical treatment should not be carried out too late.

Primary Carcinoma of the Fallopian Tube.—E. KEHRER (*Monatsschrift f. Geburtsh. u. Gynäkol.*, 1908, xxvii, 327) reports a case of carcinoma of the right tube, and reviews the 79 cases of tubal carcinoma previously reported. According to another, tubal carcinoma cannot be differentiated from pyosalpinx by location, form, size, consistency, or adhesions. For this reason a probable diagnosis of tubal carcinoma has been made only twice, and a certain diagnosis only once. A rapidly growing tumor in the cul-de-sac and a hemorrhagic exudate withdrawn through a puncture in the vaginal vault are significant. Tubal carcinoma is considered more malignant than carcinoma of the cervix or fundus, on account of the thin walls of the tube and its rich lymphatic supply. A radical operation performed as early as possible is therefore indicated. Kehrer does not believe that tubal carcinoma is necessarily preceded by inflammatory changes, as held by Sänger-Barth, and he would differentiate between papillary carcinoma of the tube and alveolar carcinoma merely as early and late stages of the same process.

Results of the Radical Abdominal Operation for Carcinoma of the Uterus.—E. WERTHEIM (*Zntrbl. f. Gynäkol.*, 1908, xxxii, 175) gives the results of his first 120 operations for carcinoma of the uterus by the method which bears his name. These operations were performed over five years ago. Of a total primary mortality of 27, 14 deaths occurred in the first 30 cases and 13 in the remaining 90. Excluding from the 93 cases in which recovery followed the operation 3 cases of carcinoma of the fundus, 1 case of carcinoma of the vulva, and 2 cases in which death was not due to cancer, Wertheim has 87 cases which have now been under observation for five years since operation. In 51 of these cases, or 58.6 per cent., there has been no return of the disease. Of 4 cases in which carcinomatous glands were removed, 3 have shown no return. Wertheim has now performed over 400 operations of this character, and without lessening in any way the thoroughness of the operation, has greatly reduced the primary mortality, so that in the last 158 cases there were only 12 deaths, or a mortality of 7.5 per cent. That the operation as performed is still radical is indicated by the fact that in

the last 158 cases necrosis of the ureter and fistula formation occurred in 10 cases. In 7 of these the fistula healed spontaneously. The improvement in technique, to which is attributed the diminished mortality, relates primarily to a better control of hemorrhage.

Phlebitis following Abdominal Operations.—O. G. PFAFF (*Amer. Jour. Obst.*, 1907, lvi, 630) states that this distressing complication occurs in 2 per cent. of all abdominal operations, and draws the following conclusions: (1) Many of these cases are simply extensive aseptic blood clots, without any true inflammation. (2) An abnormal plasticity of the blood must be present in order that thrombosis may be the result of surgical traumatism. (3) The clot generally receives a mild form of infection introduced into the wound at the time of the operation, and in turn an invasion of the vein wall results. (4) As stagnation is such an important element in the etiology, getting our patients up earlier will undoubtedly reduce the liability to thrombosis. (5) As an abnormally high degree of plasticity of the blood is essential in developing the disorder, the blood ought to be tested by some recognized standard in every case, and, if found in a dangerous state, operation should be postponed until medication shall have brought it back to a normal condition.

Neurasthenia in its Relation to Pelvic Symptoms in Women.—E. A. WEISS (*Amer. Jour. Obst.*, 1908, lvii, 230) believes that neurasthenic pelvic symptoms in women frequently exist without pathological changes in the pelvis; that a careful differentiation between neurasthenia and real morbid anatomy must be made before deciding on the treatment, and that this form of neurasthenia is not only unimproved but frequently aggravated by surgical treatment.

Protrusion of the Uterus in Nulliparous Women.—GEORGE ERETY SHOEMAKER (*Jour. Amer. Med. Assoc.*, 1907, xlix, 2151) reports two cases of protrusion of the uterus in nulliparous women and endorses the statements of other observers that heavy lifting, pelvic deformity, and the steady downward pressure of various tumors, such as ovarian dermoid cysts, are the usual causative agents. Shoemaker inveighs against radical exsections for relief of the condition mentioned.

Fibroid Operations during Pregnancy.—J. H. CARSTENS (*Amer. Jour. Obst.*, 1907, lvi, 736) reports 7 cases of operation for fibroid during pregnancy, with 1 death. One, a myomectomy case, had a very long pedicle and fibroid complicating a three months' pregnancy, and went to full term; another had fibroids complicating a five months' pregnancy; myomectomy was done, and two months later the uterus was spontaneously emptied. In a third case no result is known. In 2 others prompt abortion followed, while in the remaining case hysterectomy was done.

The Frequency and Significance of Endometritis.—B. M. ANSPACH (*Jour. Amer. Med. Assoc.*, 1908, l, 842) states that the classification of endometritis by different writers is lamentably at variance. In six text-books taken at random twenty-seven different varieties of endometritis were described, and not a single one of them was mentioned in

each book. Anspach says the presence of a discharge is in a large number of instances regarded as *prima facie* evidence of endometritis and that such practice exaggerates the importance of the endometrium in the eyes of the general practitioner. Anspach believes that but a small portion of the cases curetted for endometritis have changes in the mucosa demonstrable with the microscope. He examined the endometrial scrapings from 174 cases of dilatation, and curettage and in 80 of them the report was "normal." In 81 cases of repair or amputation of a lacerated cervix the endometrium was normal in 15; in 78 hysteromyometcomies the endometrium was normal in 11; in 60 cases of pelvic inflammatory disease the endometrium was normal in 12, and in 53 of retroposition of the uterus it was normal in 24. Anspach insists that the endometrium is not abnormal in fully half the cases diagnosticated as endometritis and offers a warning against encouraging hope of stopping leukorrhœa by curettage.

Tubal Twin Pregnancy.—CHARLES G. CHILD, JR. (*Jour. Amer. Med. Assoc.*, 1907, xlix, 2134) reports a case of tubal twin pregnancy, which he says is the sixth to be reported, the other 5 being those of Schauta, Sanitor, Cameron, McConn and Henricius, and Koester. In addition, Child says his is the first in which the fœtuses were of equal size, and the second (McConn's being the first) in which both were in one amniotic sac.

Unrecognized Gonorrhœa in the Female.—S. W. BANDLER (*Jour. Amer. Med. Assoc.*, 1908, l, 335) believes that very commonly the gonorrhœal nature of vaginal discharges and vague pelvic pains, sterility, absolute or relative, profuse menstrual flow, and chronic, mild urethritis is overlooked. Bandler believes that chronic prostatic gonorrhœa often causes chronic gonorrhœa in the female, giving rise to an entirely different clinical picture from that of acute gonorrhœa.

Final Results of Conservative Surgery of the Tubes and Ovaries.—ABRAM BROTHERS (*Jour. Amer. Med. Assoc.*, 1908, l, 595) reports having practised so-called conservative surgery of the uterine appendages in 158 cases. In but 85 of these subsequent reports are available, though Brothers states he earnestly requested patients to report to him. He frankly admits that in the presence of pus in the Fallopian tubes exsection of the tube is the preferable procedure, although ovaries apparently normal in appearance that are in juxtaposition to such tubes or pelvic pus collections can safely be left undisturbed or may be subjected to partial exsection. Brothers says, "These ovaries, if not the seat of abscess at the time of operation, seldom, in my experience, give rise to trouble later." Many gynecological surgeons will heartily commend his practice of removing by the abdominal route the pregnant Fallopian tube, although some so-called conservatives will advocate saving such structures. In considering the treatment of pus tubes Brothers states: "My practice is in favor of total and complete ablation of pus tubes down to the horn of the uterus. Experience with secondary operations for infected stumps has driven me to this position." Brothers estimates 23.5 per cent. morbidity after conservative operations on the tubes and ovaries in his practice. This estimate is based upon observation of half

his patients for periods varying from one month to twelve years. It is more than probable that the proportion of failures was much higher among the non-reporting patients than among those subsequently under observation. This statement is based largely upon the much larger proportionate number of women that apply for treatment after operations of a conservative nature done by other surgeons as compared to operations of a radical nature. The experience of many gynecologists in operations following "would-be" conservative operations by themselves and by others has driven them to abandon the "conservative" plan.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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A New Method of Entering the Maxillary Sinus through the Nose.—VAIL (*Laryngoscope*, January, 1908) penetrates the wall of the antrum with a special perforating bistoury, with which he then enlarges the cut, forward and backward, to permit the introduction of a special grooved saw, with which he saws out a large piece, making a large oval opening in the wall. In this operation sacrifice of the anterior portion of the inferior turbinate is avoided.

New Operation on Accessory Sinuses of the Nose.—WILLIAMS (*Bristol Med.-Chir. Jour.*, March, 1908) contributes an illustrated lecture on suppurative diseases of the nose and ear, with special reference to some newer methods of treatment. Williams' method, for which we have not space to detail, is an osteoplastic procedure which gives very free access to the ethmoid cells, sphenoidal sinus, and frontal sinus, and yet avoids the destruction of the nasal bone; and above all does not leave a depressed pit below the bridge, because no bridge has been made.

Trichinosis of the Upper Respiratory Passages.—MACKENTY (*Amer. Med.*, February, 1908) reports 4 cases, in 2 of which throat symptoms predominated in the form of œdema of the larynx. Three lived in the same house and the other in the same block. All of them gave a history of eating ham bought from a store in the vicinity.

Serum Therapy in Epistaxis.—SCHIFFERS (*Arch. Internat. de Laryng., d'Otol., et de Rhin.*, January and February, 1908) discusses this subject and reports 2 cases of his own, one in a hemophilic and one in a subject of essential purpura, in which the dyscrasia was successfully combated by hypodermic injections of fresh serum from the rabbit.

A Nasal Sporozoon.—JONATHAN WRIGHT (*New York Med. Jour.*, December 21, 1907) discusses, describes, and depicts the microscopic findings in a nasal sporozoön which he has studied from sections of a morbid growth sent to him by Dr. Ellett, of Memphis, which he believes to be identical with a growth of similar nature reported by Major Kinealy, of London (*Journal of Laryngology*, 1903). Wright names this parasitic protozoa, Rhinosporidium kinealyi, and states that it belongs to one of the most simple and primitive of the sporozoa.

Wounds of the Optic Nerve in Operations on the Nose.—ONODI (*Arch. internat. de laryng., d'otol., et de rhin.*, March to April, 1908) discusses the subject of wounds of the optic nerve produced by opening and emptying the posterior ethmoidal cells and the sphenoidal sinus, as well as in radical operations upon the frontal sinus and even in operations upon the septum of the nose. He attributes the result to either direct or indirect fracture of the optic canal.

Morbid Growths of the Pharynx.—MAGNE (*Rev. hebd. de laryng., d'otol., et de Rhin*, April 4, 1908) reports a case of benign growth of the size of a pigeon egg in the inferior portion of the pharynx of a man, aged sixty-three years. Under light cocainization of its pedicle the growth was readily removed without hemorrhage by the electric incandescent snare. Examination of the tumor revealed it as a fibromyxoma. Magn gives a general summary of benign growths of the buccal and inferior portions of the pharynx.

Direct Lateral Laryngoscopy and Œsophagoscopy.—MOSHER (*Boston Med. and Surg. Jour.*, February 6, 1908) places the patient flat on the back with the head turned on the left cheek; and then, usually under ether anesthesia, he uses a bivalve speculum which he has devised and which is a combination of a tongue depressor and mouth gag, made for use upon the left side so that the operator can have his right hand for the manipulation of instruments. It exposes a larger field to vision than any other device hitherto employed, and utilizes the shortest anatomical route to the larynx and to the œsophagus.

Bronchoscopy.—A. INGALS (*Jour. Amer. Med. Assoc.*, March 7, 1908), in reporting the removal of a pin fom the lung by upper bronchoscopy, refers to several severe electric shocks which he has received from the Kirstein light on his forehead while doing bronchoscopy and lighter shocks from the carrier of the small light passed through the tubes, and he has appreciated some danger in similar shocks to the patient. To prevent this he now wears rubber gloves and rubber overshoes and places his stool upon a rubber sheet. To guard the patient there are rubber castors on the table, but he thinks that we have all overlooked the possibility of the electric charge passing from the instruments through the patient and the assistants. He puts the question whether some of the hitherto inexplicable deaths from bronchoscopy may not have been due to galvanization of the vagus nerves; danger from which by instruments introduced for electrolysis of œsophageal strictures is well known to laryngologists; and in bronchoscopy the metal tube is quite as close to these nerves as the electrode would be in the electrolytic

procedure mentioned, and in unexpected ways the current may be passed through them. [It is for this reason that cautious laryngologists prefer to use the current from the battery rather than to utilize the street current when electric lights are to be passed into the interior of the body. J. S.-C.]

Papillomas of the Larynx in Young Infants.—Van Den Wildenberg (*Arch. internat. de laryng., d'otol., et de rhin.*, November and December, 1907) reports two cases of papillomas in infants, aged seventeen and eighteen months, respectively, in which the diagnosis was made by direct laryngoscopy with Killian's spatula under general anesthesia, with the head in the Rose position; the growths were removed by direct access with Killian's cutting forceps. In one case, however, cyanosis took place as a tumor was removed, and immediate tracheotomy was necessary. The next day remnants of the growth were removed by direct access as before. Both cases terminated favorably.

Sarcoma of the Mediastinum Invading the Thyroid Gland.—Snyder (*Jour. Amer. Med. Assoc.*, March 7, 1908) reports a tumor of the thyroid gland apparently connected with a mediastinal growth in a man, aged thirty-seven years, which was removed in extremis on account of the dyspnœa. The breathing, however, was only temporarily relieved and the patient died forty-five days after the operation. The autopsy revealed a large tumor occupying the entire mediastinum and adherent to all the neighboring organs except the left lung, while both lungs were completely collapsed and absolutely flat. The growth was found to be a round-cell sarcoma.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Experimental Pleurisy.—Opie (*Jour. Exp. Med.*, 1907, ix, 391) has been able to produce quite an extensive exudate into the pleural cavity of dogs by injections into this cavity of small amounts of turpentine. The pleurisy which is thus produced resembles in many respects certain forms of this disease in man, and by percussing the chest of the dog it is readily possible to follow the rapid accumulation of the exudate during the acute stages as well as its gradual disappearance. The exudate is a coagulable fluid. The serous surfaces become covered by a layer of fibrin. The acute stage lasts about six days, while the fluid reaches its height in about three days. After six days the fluid has

generally disappeared, but the fibrin, although diminished in amount, is still present. At the end of two to three weeks the pleura regains its normal state except for the presence of a few fibrinous tags. The inflammation sometimes spreads from one pleural cavity to the other, so that if turpentine is injected into the right cavity fluid might accumulate in the left side.

The most important part of the investigation is concerned with the dissolution of the fibrin. It was found that the fibrin removed from the pleural cavity during the early stages of the inflammation, after the serum had been removed by thorough washing in salt solution, undergoes digestion when it is suspended in an alkaline or in an acid fluid, whereas the fibrin taken from the cavity during the latter stages of the process, for instance after five days, fails to digest in an alkaline medium, but digests readily in an acid medium. The digestion of the fibrin outside the body may be explained by the fact that cells which contain proteolytic enzymes are present in great numbers in the meshes of the fibrin. During the first part of the inflammation, polymorphonuclear leukocytes predominate, cells which contain leukoproteose, an enzyme acting best in an alkaline medium, while in the second stage the polymorphonuclear leukocytes have disappeared and the mononuclear cells are the elements which furnish the enzyme. This enzyme acts only in an acid medium.

That digestion of fibrin takes place within the pleural cavity during the inflammatory reaction was shown by the appearance of peptone and albumoses in the pleural fluid. These bodies were found in the pleural fluid after three days, and were present in less quantity at a later time. As is true of the blood serum, so the pleural fluid was found to inhibit the action of the enzymes contained in the leukocytes during all stages of the process. The exudate remains alkaline during the entire course of the inflammation, but its alkalinity is less than the blood and decreases slightly with the progress of the inflammation.

Since the acids, which in vitro favor the actions of the enzymes present during the latter stages of the inflammation, are not found in the body, it was thought possible that carbon-dioxide might bring this enzyme into action. This supposition was found to be true, for the presence of carbon-dioxide hastened very greatly the digestion of strips of fibrin suspended in salt solution. It was found, moreover, that carbon-dioxide would overcome the inhibitory effect which blood serum has upon these cellular enzymes, so that in the presence of small quantities of blood serum, carbon-dioxide causes greater enzymotic activity than in the presence of salt solution alone.

The Combining Properties of the Opsonin of an Immune Serum.—It is known, through the work of various observers, that the opsonin present in normal blood serum is a thermolabile body which is destroyed at a temperature of 55° C. Muir and Martin have shown in a previous communication that these thermolabile opsonins of normal serum may be removed from the serum by substances or combinations of substances which absorb serum complements. This work has been extended with the same result, so that the authors are prepared to group the normal thermolabile opsonins with the complements. Observations by many experimenters have been accumulating to show that in immune sera the substance which causes phagocytosis of bacteria or red blood corpuscles

is thermostabile and resists a temperature of 55° C. for one hour. MUIR and MARTIN, (*Proceedings of the Royal Society*, B,1907, lxxix, 187) have taken up the question of the opsonins of immune sera and have studied these bodies in the sera of animals immunized against Staphylococcus aureus. They find that only a part of the opsonins of this immune serum is destroyed by a temperature of 55° C. After treating the serum with complement-absorbing substances the power of the serum to phagocyte staphylococci is only partly lost. Since both heat and complement-absorbing substance remove approximately the same amount of opsonin, Muir and Martin conclude that this portion is the normal opsonin and that the remaining thermostabile portion is a different substance, namely, the opsonin of immune serum. These immune opsonins were found, moreover, to have a certain degree of specificity. Whereas a great variety of bacteria removed large quantities of the opsonin for Staphylococcus aureus from normal serum, they removed very little of the opsonin for this particular organism from immune serum. Staphylococcus aureus will, on the other hand, absorb practically all the opsonin from immune serum. The opsonin of immune serum acts in a distinctly different manner from the opsonin of normal serum. While the normal opsonin is of the nature of the complement, the immune opsonin resembles the constitution of an agglutinin or of an immune body.

Lipase in the Blood.—It has been shown by Neuberg, Reicher, and others that bactericidal and antitoxic sera contain a ferment which is capable of splitting fats; and PRYBRAM (*Zentralbl. f. inn. Med.*, 1908, xxix, 81), with the work of these investigators in mind, undertook some experiments to determine whether a lipolytic ferment could be demonstrated in the sera of patients suffering from various diseases. The technique consisted in allowing blood serum to remain in contact with olive oil. The tube containing this mixture was placed in the thermostat at 36° C., and another tube kept as a control in the ice chest. The mixtures were titrated with $\frac{2}{10}$ normal sodium hydrate; phenolpthalein was used as an indicator. The presence of acid in the mixture kept in the thermostat showed that the olive oil had been partially converted into fatty acid. The examination of 21 cases showed that lipase was present only in the sera of those patients who had a high fever. In one case lipase could be demonstrated in the serum after the patient was exposed to *x*-rays, and therefore, to see if the *x*-rays had any influence in giving rise to this ferment in the blood serum, rabbits were exposed to the action of the *x*-rays and their serum was tested before and after treatment. These last experiments yielded varying results, and it could not be determined what action, if any, the *x*-rays played.

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